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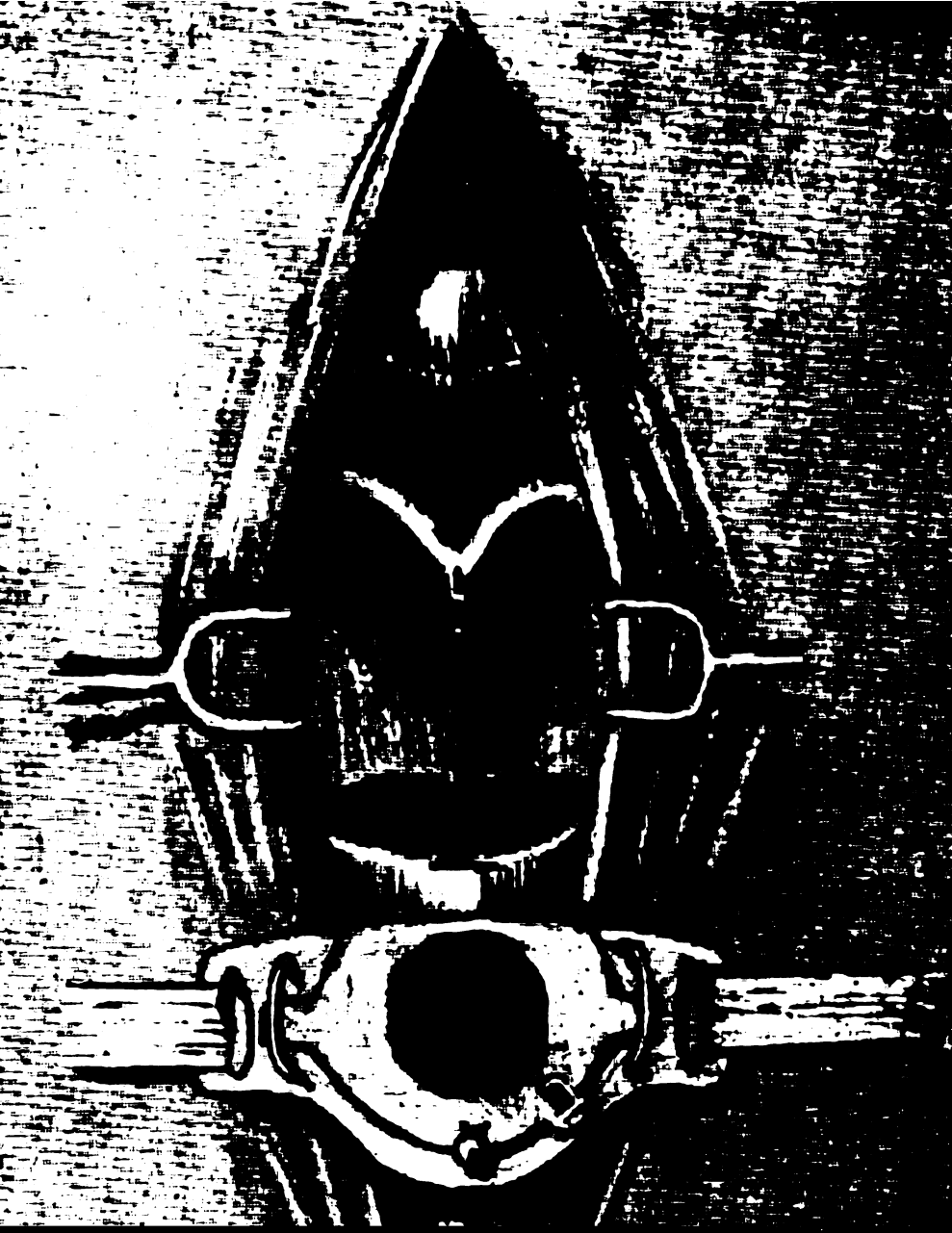

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TRANSACTIONS

OF

THE MEDICAL SOCIETY

OF

LONDON.

VOLUME THE TWENTY-FIFTH.

EDITED BY

ARTHUR FRANCIS VOELCKER, M.D., F.R.C.P.,

AND

JONATHAN HUTCHINSON, Junr., F.R.C.S.

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THE present Volume comprises the Transactions of the Society during its one hundred and twenty-ninth Session, from October 14th, 1901, to May 26th, 1902.

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1898. JOHN H. MORGAN, On the Affections of the Urinary Apparatus in Children.

1899. SAMUEL WEST, M.D., On Some of the Clinical Aspects of Granular Kidney.

1900. Sir WILLIAM MITCHELL BANKS, Practical Observations on Cancer of the Breast.

1901. J. MITCHELL BRUCE, M.D., Diseases and Disorders of the Heart and Arteries in Middle and Advanced Life.

1902. ALFRED PEARCE GOULD, On Certain Diseases of the Blood-Vessels.

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THE ORATORS.

1774. James Sims, M.D.
1776. John Millar, M.D.
1777. Nath. Hulme, M.D., F.R.S.
1778. John Coakley Lettsom, M.D., F.R.S.
1779. George Edwards, M.D.
1780. John Kooystra, M.D.
1781. Samuel Foart Simmons, M.D., F.R.S.
1782. Loftus Wood, M.D.
1783. John Sims, M.D.
1784. John Whitehead, M.D.
1785. John Relph, M.D.
1787. Joseph Hooper.
1788. John Meyer, M.D.
1789. Richard Dennison, M.D.
1790. George Wallis, M.D.
1791. Samuel Sutton, M.D.
1792. Edward Fryer, M.D.
1793. James Jameson, M.D.
1794. Gilbert Thompson, M.D.
1795. John Abernethy.
1796. John Coakley Lettsom, M.D., F.R.S.
1797. James Ware.
1798. Samuel Ferris, M.D., F.R.S.
1799. Edward Ford.
1800. Thomas Bradley, M.D.
1801. William Chamberlaine.
1802. John Sims, M.D.
1803. John Andrée.
1804. John Coakley Lettsom, M.D., F.R.S.
1805. George Pinckhard, M.D.
1806. Henry Field.
1807. Joseph Adams, M.D.
1808. John Mason Good, F.R.S.
1809. Sayer Walker, M.D.
1810. George Birkbeck, M.D.
1811. William Blair.
1812. Richard Temple, M.D.
1813. Richard Saumarez, F.R.S.
1814. George Rees, M.D.
1815. John Taunton.
1816. Henry Clutterbuck, M.D.
1817. John Stevenson.
1818. David Uwins, M.D.
1819. Thomas J. Pettigrew, F.R.S.
1820. Thomas Hancock, M.D.
1821. Thomas Callaway.
1822. James Copland, M.D.
1823. Edward Grainger.
1824. Gordon Smith, M.D.
1825 Eusebius Arthur Lloyd.
1826. John Haslam, M.D.
1827. William Kingdom.
1828. John Burne, M.D.
1829. William Greville Jones.
1830. Leonard Stewart, M.D.
1831. Montague Gossett.
1832. John Whiting, M.D.
1833. Frederick Salmon.
1834. William Shearman, M.D.
1835. Walter Cooper Dendy.
1836. William F. Blicke, M.D.
1837. Edward Headland.
1838. Theophilus Thompson, M.D., F.R.S.
1839. George Pilcher.
1840. James Risdon Bennett, M.D.
1841. Wm. Dingle Chowne, M.D.
1842. Henry Hancock.
1843. Leonard Stewart, M.D.
1844. Thomas Bell, F.R.S.
1845. Marshall Hall, M.D.
1846. John Bishop, F.R.S.
1847. Golding Bird, M.D., F.R.S.
1848. Francis Hird.
1849. William Hughes Willshire, M.D.
1850. Francis Hird.
1851. Richard Rowland.
1852. Edwin Canton.
1853. John Snow, M.D.

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1854. Henry Smith.
1855. James Fernandez Clarke.
1856. Benj. Ward Richardson, M.D., F.R.S.
1857. William Adams.
1858. Alfred Baring Garrod, M.D.
1859. Charles Henry Felix Routh, M.D.
1860. John Gay.
1861. Arthur Leared, M.D.
1862. Victor de Méric.
1863. Samuel Osborne Habershon, M.D.
1864. John Louis William Thudichum, M.D.
1865. Robert Greenhalgh, M.D.
1866. Thomas Christopher Weeden Cooke.
1867. Frederick William Headland, M.D.
1868. William Frederick Teevan.
1869. George Duncan Gibb, M.D.
1870. Francis Mason.
1871. William Cholmeley, M.D.
1872. Frederick James Gant.
1873. John Cockle, M.D.
1874. Robert Brudenell Carter.
1875. George Buchanan, M.D.
1876. Erasmus Wilson, F.R.S.
1877. John Hughlings Jackson, M.D., F.R.S.
1878. Alfred Carpenter, M.D.
1879. Walter John Coulson.
1880. William Henry Broadbent, M.D.
1881. Arthur Edward Durham.
1882. Edmund Symes Thompson, M.D.
1883. Edward Lund.
1884. Charles Theodore Williams, M.D.
1885. George Murray Humphry, M.D., F.R.S.
1886. Richard Douglas Powell, M.D.
1887. Sir William MacCormac.
1888. Sir Joseph Fayrer, K.C.S.I., M.D., F.R.S.
1889. Jonathan Hutchinson, F.R.S.
1890. Arthur Ernest Sansom, M.D.
1891. Sir Joseph Lister, Bart., F.R.S.
1892. Sir James Crichton-Browne, M.D., F.R.S.
1893. W. Mitchell Banks.
1894. William M. Ord, M.D.
1895. A. Pearce Gould.
1896. William H. Allchin, M.D.
1897. Edmund Owen.
1898. Frederick T. Roberts, M.D.
1899. Alban Doran.
1900. J. Kingston Fowler, M.D.
1901. F. Richardson Cross.
1902. Stephen Mackenzie, M.D.

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THE FOTHERGILLIAN GOLD MEDALLISTS.

1787. William Falconer, M.D.
1790. Robert Willan, M.D.
1791. John Coakley Lettsom, M.D.
1795. John Mason Good.
1801. Francis Bouttatz, M.D.
1803. Edward Jenner, M.D.
1824. Robert W. Bampfield.
1828. John George Parry.
1831. William Augustus Guy.
1834. William James Clement.
1835. George Moore.
1836. Thomas Egerton Bryant.
1838. George Pilcher.
1840. Samuel Osborn.
1842. James Risdon Bennett, M.D.
1843. John Weaver Lever, M.D.
1844. Henry Pratt Roberts.
1845. Walter Cooper Dendy.
1846. Robert Mortimer Glover, M.D.
1847. Silas Stedman.
1849. John Milligan.
1850. Richard Payne Cotton, M.D.
1851. Richard Hodges.
1852. Frederick William Headland.
1853. Alfred William Poland.
1854. Benjamin Ward Richardson, M.D.
1856. William Burke Ryan.
1857. Edwin Canton.
1858. Thomas Herbert Barker M.D.
1859. Alderman Thomas Houghton Waters.
1868. John Clay.
1870. Thos. Smith Clouston, M.D.
1872. Edwards Crisp, M.D.
1873. John Kent Spender, M.D.
1877. Peter Murray Braidwood, M.D.
1878. John Milner Fothergill, M.D.
1882. Thomas Michael Dolan, M.D.
1883. Norman Porritt.
1886. John Strahan.
1888. Hobart Amory Hare, M.D., U.S.A.
1893.* William Richard Gowers, M.D., F.R.S.
1896. Victor Horsley, F.R.S.
1899. Sydney Monckton Copeman, M.D.
1902. Patrick Manson, C.M.G., M.D., F.R.S.

* *First Triennial Award under the Charity Commissioners' New Scheme,* vide *Trans., vol. xiv, p. xi.*

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THE HONORARY FELLOWS.

1893. BARNES, ROBERT, M.D., Bernersmede, Eastbourne, LL, C.

1896. BERGER, PAUL, 16, Rue de Bourgogne, Paris. Professor of Clinical Surgery in the Faculty of Medicine, Member of the Academy of Medicine, Surgeon to "l'hôpital de la Pitié."

1897. BERGMANN, Prof. E. VON, M.D., Berlin.

1881. BILLINGS, JOHN S., M.D., Washington, Surgeon to the United States Army.

1899. BROUARDEL, PAUL CAMILLE HIPPOLYTE, 85, Boulevard St. Germains, Paris. Professor of Medical Jurisprudence, late Dean of the Faculty of Medicine, University of Paris; Member of the Institute and of the Academy of Medicine.

1894. CATRIN, LOUIS, M.D., Médecin-major de première classe, Professeur agrégé à l'Ecole du Val-de-Grace, Paris.

1873. CHAUVEAU, A., late Professor of Physiology at the Medical School of Lyons, 10, Avenue Jules Janin, Paris.

1881. EMMET, THOMAS ADDIS, M.D., 89, Madison-avenue, Surgeon to the Woman's Hospital of the State of New York.

1899. FAYRER, Sir JOSEPH, Bart., K.C.S.I., LL.D., M.D., F.R.S., 16, Devonshire-street, Portland-place, W. P, VP, LL, SM, O, C.

1886. GAIRDNER, Sir WILLIAM TENNANT, K.C.B., M.D., F.R.S., 32, George-square, Edinburgh, formerly Professor of Medicine in the University of Glasgow, Physician to H.M. the King in Scotland.

1894. GANT, FREDERICK JAMES, F.R.C.S., 16, Connaught-square, W., Consulting Surgeon to the Royal Free Hospital. P, VP 2, LL, O, C 3.

1897. GUYON, FELIX, M.D., Professor in the Faculty of Medicine, Member of the Institute and of the Academy of Medicine, Paris.

1897. HINGSTON, Sir W. HALES, M.D., Montreal.

1890. KOCHER, THEODOR, Professor, Berne.

1878. MITCHELL, S. WEIR, M.D., Walnut-street, Philadelphia.

1899. OSLER, WILLIAM, M.D., F.R.S., Baltimore, U.S.A., Professor of Medicine, Johns Hopkins University, and Physician-in-Chief, Johns Hopkins Hospital.

1899 RAUCHFUSS, CHARLES ANDREIVITCH, 8, Ligovka, St. Petersburg, Director of the Prince of Oldenburg's Hospital for Children.

1897. RODDICK, T. G., M.D., 80, Union-avenue, Montreal.

1877. SANNÉ, A., 33, Rue de Naples, Paris.

1899. SKLIFOSOFFSKY, NICHOLAS VASILIVITCH, M.D., Director and Professor of Surgery, Imperial Clinical Institute of the Princess Helena Paulovna, St. Petersburg.

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CORRESPONDING FELLOWS.

1882. Badaloni, Giuseppe, M.D., Bologna, Italy.
1856. Baker, Albert, M.D., Fosse Cottage, Batheaston, Bath.
1850. Beneke, F. W., M.D., New York.
1850. Böhm, Professor, M.D., Vienna.
1850. Bottini, Enrico, M.D., Milan.
1855. Coates, Charles, M.D., F.R.C.P., 10, Circus, Bath, Consulting Physician to the Bath Royal United Hospital. c 3.
1850. Cox, William Isidore, Hawkesbury-Upton, Gloucestershire. c.
1853. Fallot, R., M.D., St. Laurent d'Aigonze, Montpelier, France.
1889. Frank, Philip, M.D., F.R.C.P., 3, Elvaston-place, S.W.
1876. Griffith, Richard Glyn, M.R.C.S., Portledge House, Bath.
1896. Hamilton, Allan McLane, M.D., 44, East Twenty-ninth Street, New York.
1864. Hasenfeld, Emmanuel, M.D., Pesth.
1875. Jones, Philip Sydney, M.D., F.R.C.S., 16, College-street, Sydney, Examiner in Medicine in the University of Sydney, Australia, Hon. Consulting Surgeon to the Sydney Hospital.
1851. Kölliker, Albert, M.D., Professor of Anatomy and Physiology at the University of Wurzburg.
1876. Leighton, Walter H., M.D., Lowell, Massachusetts, U.S.A.
1851. Negri, Gaetano, M.D. Pisa.
1865. Peruzzi, Domenico, M.D., 22, Via Mazzini, Bologna.
1886. Rocha, A., M.D., Professor of Clinical Medicine at the University of Coimbra, Beira, Portugal.
1860. Roussel, M.D., Dean of the Faculty of Medicine, Montpelier.
Scharlan, Gus. W., M.D., Stettin, Prussia.
1876. Schmitz, Richard, M.D., Neuenahr.
1874. Schutgowsky, J., St. Petersburg.
1851. Williams, Charles, F.R.C.S. Edin., 48, Prince of Wales-road, Norwich; Senior Surgeon to the Norfolk and Norwich Hospital.

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THE FELLOWS
OF
THE MEDICAL SOCIETY OF LONDON.

(Corrected to 8th November, 1902.)

EXPLANATION OF ABBREVIATIONS.

P.—PRESIDENT.
VP.—VICE-PRESIDENT.
T.—TREASURER.
L.—LIBRARIAN.
S.—SECRETARY.
C.—COUNCILLOR.
LL.—LETTSOMIAN LECTURER.
FM.—FOTHERGILLIAN GOLD MEDALLIST.
SM.—SILVER MEDALLIST.
O.—ORATOR.
CFC.—CHAIRMAN, HOUSE AND FINANCE COMMITTEE.
§—SEC. FOR FOREIGN CORRESPONDENCE.
*—LIFE MEMBER.
TR.—TRUSTEE.

The date prefixed is that of election. The figures appended indicate the number of Sessions served, and refer to past appointments ONLY.

1890. ABBOT-ANDERSON, WILLIAM MAURICE, M.B., 36, Harley-street, W.
1888. ABBOTT, CHARLES EDWARD, M.R.C.S., 1, Wellington-place, Cheltenham.
1891. ABRAHAM, PHINEAS S., M.D., 2, Henrietta-street, Cavendish-square, W.
1896. ABRAHAMS, BERTRAM LEWIS, M.B., 14, Welbeck-street, W.
1894. ACHARD, ALEXANDER LOUIS, M.D., 9, Blandford-street, Portman-square, W.
1890. ACKLAND, ROBERT CRAIG, M.R.C.S., 54, Brook-street, Grosvenor-square, W.
1883. ACLAND, THEODORE DYKE, M.D., 19, Bryanston-square, W. C 2.
1878. *ALLCHIN, WILLIAM HENRY, M.D., F.R.S.E., 5, Chandos-street, Cavendish-square, W. P, SM, L 18, VP 2, O, C 2. *Councillor.*
1873. ALLEN, HENRY MARCUS, F.R.C.P. Edin., 17, Palmeira-square, Brighton.
1873. ALLFREY, CHARLES HENRY, M.D., Plas Newydd, St. Leonards-on-Sea.
1883. ALLINGHAM, HERBERT W., F.R.C.S., 25, Grosvenor-street, W. C 3.
1872. *ALLINGHAM, WILLIAM, F.R.C.S., Kingsdene, Shelley-road, Worthing. C.
1894. ALSTON, WILLIAM EVELYN, M.B., 22, Bentinck-street, Cavendish-square, W.
1885. ANDERSON JOHN, C.I.E., M.D., 9, Harley-street, W. C 3.
1869. ARMITAGE, SAMUEL HARRIS TATHAM, M.D., 39, Grosvenor-street, W.
1902. ARMOUR, DONALD J., F.R.C.S., 3, Bentinck-mansions, Bentinck-street, W.
1894. ARMSTRONG, WILLIAM, M.R.C.S., J.P., Thorncliffe, Buxton.

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1894. ASHE, WILLIAM PERCY, M.D., 17, Alexander-square, S.W.
1873. ATKINSON, EDWARD, M.R.C.S., 93, Albion-street, Leeds.
1899. AULD, ALEXANDER GUNN, M.D., 23, Henrietta-street, W.
1892. AYRES, CHARLES JAMES, M.D., 55A, Welbeck-street, W.

1873. BAGSHAWE, FREDERIC, M.D., 35, Warrior-square, St. Leonards-on-Sea. C.
1892. BAILEY, HENRY FREDERICK, M.R.C.S., The Hollies, Lee-terrace, Lee, S.E. C 2.
1894. BAILEY, ROBERT COZENS, M.S., 21, Welbeck-street, W.
1891. BAILY, PERCY J., M.B., County Asylum, Hanwell, W.
1898. BAIN, WILLIAM, M.D., Shaythorpe, York-place, Harrogate.
1876. *BAKER, HENRY FRANCIS, F.R.C.S. Edin., 2, Mandeville-place, Manchester-square, W. C.
1890. BAKER, WILLIAM HENRY, M.R.C.S., 152, Westbourne-grove, W. C 2.
1891. BALL, JAMES BARRY, M.D., 12, Upper Wimpole-street, W.
1881. BALLANCE, CHARLES ALFRED, M.S., 106, Harley-street, W. S 2, C 3. *Vice-President.*
1884. BANKS, Sir W. MITCHELL, F.R.C.S., LL.D., 28, Rodney-street, Liverpool. VP 2, O, C, LL. *Councillor.*
1899. BARKER, ARTHUR E. J., F.R.C.S., 87, Harley-street, W. *Councillor.*
1901. BARLOW, H. CECIL, M.B., Sa Maison, Somersham, Hunts.
1883. *BARNES, ROBERT, M.D., *Honorary Fellow* (*q. v.*).
1874. BARRETT, HOWARD, M.R.C.S., 49, Gordon-square, W.C. VP 2, C 2.
1896. BARRETT, WALTER RUSSELL, M.R.C.S., 1, Harley-street, W.
1901. BARRON, HENRY T., M.D., 12, Crockerton-road, Upper Tooting, S.W.
1902. BARRON, WILLIE NETTERVILLE, M.R.C.S., Cranborne Corner, Ascot.
1884. BARROW, ALBERT BOYCE, F.R.C.S., 8, Upper Wimpole-street, W. C.
1900. BARTER, WILLIAM, M.D., 47, Greencroft-gardens, West Hampstead, N.W.
1901. BARTON, PERCY FREDERIC, M.B., "The Rowans," Lingfield-road, Wimbledon, S.W.
1886. BARWELL, RICHARD, F.R.C.S., 55, Wimpole-street, W.
1886. BATTERHAM, JOHN WILLIAMS, M.B., Bank House, Grand-parade, St. Leonards-on-Sea.
1888. BATTLE, WILLIAM HENRY, F.R.C.S., 49, Harley-street, W. C 3, S 2.
1896. BAYLISS, RICHARD ARTHUR, M.R.C.S., 5, Gay-street, Bath.
1882. BEACH, FLETCHER, M.B., Winchester House, Kingston-hill, Surrey, and 79, Wimpole-street, W. C.
1887. BEALE, EDWIN CLIFFORD, M.B., 23, Upper Berkeley-street, W. C 2.
1891. BEALE, PEYTON T. B., F.R.C.S., 61, Grosvenor-street, W.
1880. BEEVOR, CHARLES EDWARD, M.D., 135, Harley-street, W. S 2, C. *Trustee.*
1889. BEEVOR, Sir HUGH REEVE, Bart., M.D., 17, Wimpole-street, W.
1900. BELFRAGE, S. HENNING, M.B., 1, Montagu-place, Portman-square, W.
1900. BELL, JAMES VINCENT, M.D., Star Hill, Rochester.
1883. BENNETT, Sir WILLIAM HENRY, K.C.V.O., F.R.C.S., 1, Chesterfield-street, Mayfair, W. C. *Orator.*

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1900. Benson, Alfred, M.B., 79, Maida Vale, W.
1896. Berkeley, Comyns, M.B., 53, Wimpole-street, W.
1887. Berry, James, F.R.C.S., 21, Wimpole-street, W. c.
1898. Best, William Harris, L.S.A., Ilford, Essex.
1873. Beveridge, James Spowart, M.R.C.P. Edin., Melton Constable, Norfolk.
1890. Bidwell, Leonard Arthur, F.R.C.S., 15, Upper Wimpole-street, W.
1888. Bird, Matthew Mitchell, M.D., St. Mary's Hospital, W.
1850. *Birkett, John, F.R.C.S., 1, Sussex-gardens, W. vp, c 6.
1889. Bisshopp, Francis Robert Bryant, M.B., Belvedere, Lonsdale-gardens, Tunbridge Wells.
1886. *Black, William Gall, F.R.C.S., 2, George-square, Edinburgh.
1897. Blacker, George Francis, M.D., 11, Wimpole-street, W.
1901. Black-Milne, James, M.B., Brookfield, Reigate.
1881. Blaker, Walter Campbell, M.R.C.S., Royal Societies Club, 63, St. James's-street, S.W.
1888. Blanc, Leon, M.D., Aix les Bains, France.
1892. Bland-Sutton, John, F.R.C.S., 47, Brook-street, W. c. *Councillor.*
1871. *Bloxam, John Astley, F.R.C.S., 75, Grosvenor-street, W. vp 2, s 2, c 3.
1879. Bott, Henry, M.R.C.S., Brentford, Middlesex.
1886. Bourns, Newcome Whitelaw, M.D., 78, Redcliffe-gardens, S.W.
1886. Boustead, Robinson, M.D., Lieutenant-Colonel, R.A.M.C., c/o Messrs. King & Co., 45, Pall Mall, S.W.
1895. Bower, David, M.D., Springfield House, Bedford.
1889. Bowles, Robert Leamon, M.D., 16, Upper Brook-street, W. c 3.
1895. Boyd, Stanley, F.R.C.S., 134, Harley-street, W. c 2.
1899. Bradford, J. Rose, M.D., F.R.S., 8, Manchester-square, W.
1896. Bradley, James Edward Campbell, M.B., Greville House, Raynes Park, Wimbledon.
1889. Braine, C. Carter, F.R.C.S., 26, Wimpole-street, W.
1876. Brewer, Alexander Hampton, M.R.C.S., 136, Richmond-road, Dalston, N.E.
1896. Bridges, E. Chittenden, M.B., Priory-mansions, Drayton-gardens, South Kensington, S.W.
1873. Bridgwater, Thomas, M.B., LL.D., J.P., Harrow, Middlesex.
1893. Broadbent, John Francis Harpin, M.B., 35, Seymour-street, W.
1862. *Broadbent, Sir William Henry, Bart., K.C.V.O., M.D., F.R.S., 84, Brook-street, W. p, vp, o, ll, c 4.
1900. Broadbent, Walter, M.D., 50, Brunswick-square, Brighton.
1890. Brook, William Frederick, F.R.C.S., Longlands House, St. Helen's-road, Swansea.
1898. Brooke, Gilbert E., L.R.C.P., East Harbour, Turk's Island, West Indies.
1878. Brown, Andrew, M.D., 27, Lancaster-road, Belsize-park, N.W.
1889. Browne, George Buckston, M.R.C.S., 80, Wimpole-street, W. c 2.
1901. Browne, Oswald A., M.D., 7, Upper Wimpole-street, W.

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1887. Bruce, John Mitchell, M.D., 23, Harley-street, W. LL, VP 2, C 3.
1873. Brunjes, Martin, M.R.C.S., 270, Willesden-lane, N.W.
1898. Brunton, John, M.B., 16, Endsleigh-street, Tavistock-square, W.C.
1874. *Brunton, Sir T. Lauder, M.D., F.R.S., 10, Stratford-place, W. LL, VP, C 4, SM.
1901. Bryant, John H., M.D., 4, St. Thomas's-street, S.E.
1850. *Bryant, Thomas, F.R.C.S., 27, Grosvenor-street, W. P, VP, LL, S 2, C 4. *Trustee.*
1901. Buckley, Charles W., M.D., 14, Hardwick-street, Buxton, Derbyshire.
1897. Bunch, John Le Mare, M.D.
1885. *Bunny, J. Brice, M.R.C.S., Warre House, Bishops Lydeard, Taunton.
1886. Butler-Smythe, Albert Charles, F.R.C.S., 76, Brook-street, W.

1886. Cahill, John, M.D., 12, Seville-street, Lowndes-square, Hyde Park, S.W. C 3.
1892. Caldwell, Robert, F.R.C.S., Major, R.A.M.C., care of Messrs. Holt and Co., 17, Whitehall-place, S.W.
1893. Caley, Henry Albert, M.D., 24, Upper Berkeley-street, W. C 3. *Hon. Secretary.*
1891. Calvert, James, M.D., St. Bartholomew's Hospital, E.C. S 2, C 2.
1897. Cameron, Ewan Gordon, M.R.C.S., 64, Great Portland-street, W.
1892. Cargill, Lionel Vernon, F.R.C.S., 31, Harley-street, W.
1892. Carless, Albert, F.R.C.S., 10, Welbeck-street, W.
1882. Carpenter, Arthur Bristowe, M.B., Wykeham House, Bedford Park, Croydon.
1889. Carr, John Walter, M.D., 19, Cavendish-place, W. C. *Councillor.*
1897. Carré, L. J. Gerard, M.D., care of Principal Medical Officer, Cape Town, South Africa.
1901. Carson, Herbert William, F.R.C.S., 26, Welbeck-street, W.
1871. Carter, Robert Brudenell, F.R.C.S., 101, Harley-street, W. P, VP, O, LL, C 4.
1889. Cartwright, Alexander, M.R.C.S., 32, Old Burlington-street, W.
1876. Cartwright, S. Hamilton, *Travelling.*
1899. Cathcart, George Clark, M.B., 35, Harley-street, W.
1876. *Cathcart, Samuel, M.R.C.P. Edin., 11, Hornsey-lane-gardens, Highgate, N.
1889. Cautley, Edmund, M.D., 15, Upper Brook-street, W.
1896. Chapman, Charles William, M.D., 21, Weymouth-street, W. *Councillor.*
1885. Chasseaud, William, M.D., Smyrna, Asia Minor.
1889. Cheyne, Watson, C.B., F.R.C.S., F.R.S., 75, Harley-street, W. LL, C.
1877. *Chisholm, Edwin, M.D., 82, Darlinghurst-road, Sydney, New South Wales.
1895. Christmas, Robert William Samuel, M.R.C.S., 23, Hopton-road, Streatham, S.W.
1871. Churton, Thomas, M.D., 35, Park-square, Leeds. C.

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1875. CLARK, ANDREW, F.R.C.S., 71, Harley-street, W. C.
1883. CLARKE, WILLIAM BRUCE, F.R.C.S., 51, Harley-street, W. C.
1894. CLINCH, THOMAS ALDOUS, M.D., 16, Regent-street, Nottingham.
1879. *CLUTTON, HENRY HUGH, F.R.C.S., 2, Portland-place, W. VP 2, C 3.
1899. COBB, ERNEST HERBERT, M.R.C.S., Belmont, Stevenage, Herts.
1894. COCHRANE, JAMES MACKEAND, M.D., 10, Weymouth-street, W.
1899. COHEN, GEORGE ALEXANDER, M.B., 17, Grand-parade, Harringay, N.
1893. COLE, ROBERT HENRY, M.D., Moorcroft, Hillingdon, Uxbridge.
1902. COLLIER, HORACE STANSFIELD, F.R.C.S., 57A, Wimpole-street, W.
1887. COLLIER, WILLIAM, M.D., St. Mary's Entry, Oxford. C.
1892. COLMAN, WALTER S., M.D., 9, Wimpole-street, W. C.
1871. COOK, JOHN, M.D., 1, Nottingham-terrace, Regent's Park, N.W.
1862. *COOPER, Sir ALFRED, F.R.C.S., 9, Henrietta-street, Cavendish-square, W. C 3, VP.
1888. COOPER, ARTHUR, M.R.C.S., 20, Old Burlington-street, W.
1894. COOPER, PETER, M.R.C.S., 35, Shooters-hill-road, Blackheath, S.E.
1894. CORBOULD, VICTOR A. L. E., M.D., 43, Victoria-road, Kensington, W.
1872 CORFIELD, WILLIAM HENRY, M.D., 19, Savile-row, W. C.
1899. CORNER, HARRY, M.D., Brook House, Southgate, N.
1891. COUMBE, JOHN BATTEN, F.R.C.S., 55, High-street, Lowestoft.
1889. COURTNEY, GUY BUDD, M.B., 28, Augusta-road, Ramsgate.
1873. CRAVEN, Sir ROBERT MARTIN, F.R.C.S., J.P., 14, Albion-street, Hull.
1889. CRAWFORD, JAMES, M.D.
1899. CREASY, L. ELIOT, M.R.C.S., 35A, Welbeck-street, W.
1871. CRICHTON-BROWNE, Sir JAMES, M.D., F.R.S., 61, Carlisle-place-mansions, S.W. P, O, C 2.
1881. CRIPPS, WILLIAM HARRISON, F.R.C.S., 2, Stratford-place, W. C 2.
1880. CRITCHETT, Sir ANDERSON, F.R.C.S. Edin., 21, Harley-street, W.
1880. CROCKER, HENRY RADCLIFFE, M.D., 121, Harley-street, W. C 3. *Lettsomian Lecturer.*
1901. CROMBIE, ALEXANDER, M.D., Lieutenant-Colonel, I.M.S. (*retired*), 3A, Bickenhall-mansions, Gloucester-place, W.
1881. CROSS, FRANCIS RICHARDSON, F.R.C.S., Worcester House, Clifton, Bristol. O.
1890. CULLINGWORTH, CHARLES JAMES, M.D., 14, Manchester-square, W. C 3.
1874. CUMBERBATCH, ALPHONSO ELKIN, F.R.C.S., 80, Portland-place, W. C 2.

1897. DAKIN, WILLIAM RADFORD, M.D., 8, Grosvenor-street, W. C.
1894. DALBY, JOHN LYTTLETON, M.R.C.S., 13, Buckingham-road, Brighton.
1871. DALBY, Sir WILLIAM BARTLETT, F.R.C.S., 18, Savile-row, W. P, C 2.
1881. DALLAWAY, DENNIS JOSEPH WILLIAM, L.R.C.P. Edin., Windham Club, St. James's-square, S.W. C 2.
1901. DANIEL, PETER LEWIS, F.R.C.S., 5, Devonshire-street, W.
1890. DAVIS, HENRY, M.R.C.S., 60, Queen Anne-street, W.

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1889. *Davison, James, M.D., "Streate Place," Bath-road, Bournemouth.
1880. Davson, Smith Houston, M.D., Campden Villa, 203, Maida-vale, W. c 3.
1868. *Davy, Richard, F.R.C.S., F.R.S.E., Burstone House, Bow, North Devon. vp, s 2, sm, § 2.
1876. Dawes, Richard St. Mark, M.R.C.S., Gawler, South Australia.
1897. Dawson, Bertrand, M.D., 110, Harley-street, W.
1880. Dawson, Yelverton, M.D., Heathland, Southbourne-on-Sea, Christchurch, Hants.
1894. Dean, Henry Percy, M.S., 69, Harley-street, W.
1883. Dent, Clinton Thomas, F.R.C.S., 61, Brook-street, Grosvenor-square, W. c 3.
1897. Des Vœux, Harold A., M.D., 14, Buckingham-gate, S.W.
1902. Dillon, Henry Vincent McMahon, L.R.C.P. and S.I., 2, Scarsdale-villas, Kensington, W.
1891. Diver, Ebenezer, M.D., Alverstoke, Gosport.
1893. Dockrell, Morgan, M.D., 9, Cavendish-square, W.
1885. Dodd, Henry Work, F.R.C.S., 136, Harley-street, W.
1882. Dolan, Thomas Michael, M.D., Horton House, Halifax. fm 1882.
1881. Doran, Alban Henry Griffiths, F.R.C.S., 9, Granville-place, W. c 4, o, § 3.
1898. Douglas, John L., M.D., 42, Central-hill, Norwood, S.E.
1890. Douglas, William, M.D., 4, St. Alban's-place, Regent-street, S.W.
1897. *Drage, Lovell, M.D., Hatfield, Herts.
1886. Duckworth, Sir Dyce, M.D., 11, Grafton-street, Piccadilly, W. c.
1884. Duncan, William, M.D., 6, Harley-street, W.
1884. Durham, Frederic, F.R.C.S., 52, Brook-street, W. c. *Councillor.*

1895. Ealand, Hugh Fawsit, L.R.C.P. Edin., The Moat, Puckeridge, Herts.
1896. Earle, Walter George, M.R.C.S., 44, Upper Grosvenor-road, Tunbridge Wells.
1899. Eastes, George Leslie, M.B., 62, Queen Anne-street, W.
1891. Eastes, Thomas, M.D., 3, Shakespeare-terrace, Folkestone.
1895. Eccles, William McAdam, M.S., 124, Harley-street, W.
1892. Eddowes, Alfred, M.D., 28, Wimpole-street, W.
1860. *Edmunds, James, M.D., 4, Chichester-terrace, Kemp Town, Brighton.
1880. Edwards, Frederick Swinford, F.R.C.S., 55, Harley-street, W. c 3.
1897. Elgood, Charles Reginald, M.D., 8, Clarence-villas, Windsor.
1901. Elliot, Andrew, M.D., 11, Upper Berkeley-street, W.
1899. Elliot, Norman Bruce, M.D., 27, Warwick-square, S.W.
1868. Elliott, George Frederick, M.D., 1, Albion-street, Hull.
1882. Elliott, Thomas, M.D., Monson-place, Tunbridge Wells.
1883. English, Edgar, M.R.C.S., St. Mary's-road, Doncaster.
1880. English, Thomas Johnston, M.D., 128, Fulham-road, S.W.
1889. Esler, Robert, M.D., 4, Queen's-road, Peckham, S.E.
1899. Evans, J. Morgan, M.R.C.S., J.P., Llandrindod Wells, Radnorshire.

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1894. EVANS, WILLMOTT H., F.R.C.S., 2, Upper Wimpole-street, W.
1893. EVE, FREDERIC S., F.R.C.S., 125, Harley-street, W.
1883. EWART, Sir JOSEPH, M.D., J.P., Dep. Surgeon-General, Bengal Army (*retired*), Bewcastle, Dyke-road, Brighton. c.
1877. *EWART, WILLIAM, M.D., 33, Curzon-street, Mayfair, W. c 3, § 3. *Councillor.*

1902. FAIRBANK, HAROLD ARTHUR THOMAS, F.R.C.S., 23, Porchester-gardens, Hyde-park, W.
1884. FARDON, EDWARD ASHBY, M.R.C.S., Middlesex Hospital, W.
1873. *FAYRER, Sir JOSEPH, Bart., K.C.S.I., LL.D., M.D., F.R.S., *Honorary Fellow* (*q. v.*).
1896. FAYRER, JOSEPH, M.D., Captain, R.A.M.C., India.
1898. FELKIN, ROBERT WILLIAM, M.D., 48, Westbourne-gardens, Bayswater, W.
1884. FENTON, FREDERICK ENOS, F.R.C.S., 33, Welbeck-street, W.
1885. FENWICK, EDWIN HURRY, F.R.C.S., 14, Savile-row, W. c.
1887. FERRIER, DAVID, M.D. Edin., F.R.S., 34, Cavendish-square, W. c.
1878. FIELD, GEORGE, M.R.C.S., 34, Wimpole-street, W. c.
1894. FISHER, FREDERICK CHARLES, F.R.C.S., King's Langley, Herts.
1876. FISHER, FREDERICK RICHARD, F.R.C.S., 18, Wimpole-street, W.
1899. FLETCHER, H. MORLEY, M.D., 98, Harley-street, W.
1884. FLINT, ARTHUR, M.D., J.P., Westgate Lodge, Westgate-on-Sea.
1878. *FONMARTIN, HENRY DE, M.D., 26, Newberry-terrace, Lower Bullar-street, Southampton.
1893. FORBES, NORMAN HAY, F.R.C.S. Edin., J.P., "Drumminor," Tunbridge Wells.
1899. FORD, FRANK CHUBB, M.B., 1, Sunnyside, Wimbledon, S.W.
1879. FOWLER, JAMES KINGSTON, M.D., 35, Clarges-street, Mayfair, W. s 2, c 2, vp 2, o.
1873. FOX, ARTHUR EDWARD WELLINGTON, M.B., C.M., Ennox Lodge, Hinton, Charterhouse, near Bath. c.
1887. FOX, FORTESCUE, M.D., Strathpeffer Spa, Ross-shire.
1871. FOX, FRANCIS, M.R.C.S., 15, Arundel-gardens, Kensington Park, W. c 3.
1885. *FOX, R. HINGSTON, M.D., 20, Gordon-square, W.C.
1879. FOX, THOMAS COLCOTT, M.B., 14, Harley-street, W. s 2, c 2.
1868. FREER, ALFRED, M.R.C.S., J.P., Stourbridge, Worcestershire.
1896. FREYER, P. JOHNSTON, M.Ch., Surgeon-Lieut.-Colonel (*retired*), 46, Harley-street, W.
1886. FRITH, BAPTIST GAMBLE, M.B., Drumgay, Grove-road, Guildford.
1884. FROST, WILLIAM ADAMS, F.R.C.S., 30, Cavendish-square, W. c 2.

1895. GALLOWAY, JAMES, M.D., 54, Harley-street, W.
1862. *GANT, FREDERICK JAMES, F.R.C.S., *Honorary Fellow* (*q. v.*).
1899. GARDENER, WILLIAM FREDERICK, M.R.C.S., Darley House, Venner-road, Sydenham, S.E.
1847. *GARROD, Sir ALFRED BARING, M.D., F.R.S., 10, Harley-street, W. p, vp 2, ll, o, c 9.

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1893. Garson, John George, M.D., 14, Stratford-place, W.
1891. Gaster, Aughel, M.D., 68, Greencroft-gardens, South Hampstead, N.W.
1887. Gay, John, M.R.C.S., 119, Upper Richmond-road, Putney, S.W.
1856. *Gibbon, Septimus, M.B., 39, Oxford-terrace, W.
1882. Gibbons, Robert Alexander, M.D., 29, Cadogan-place, S.W. c. *Councillor.*
1893. Gibson, Charles, M.D., 5, Beech-grove, Harrogate.
1897. Gibson, George Alexander, M.D., 3, Drumsheugh-gardens, Edinburgh.
1881. Giffard, Douglas W., M.R.C.S., 5, Pavilion-parade, Brighton.
1894. Gill, Richard, F.R.C.S., 72, Wimpole-street, W.
1867. Gill, William, M.R.C.S., 11, Russell-square, W.C. c.
1900. Godfrey, Thomas Henry, M.B., The Red House, Church-end, Finchley, N.
1869. *Godson, Clement, M.D., 82, Brook-street, W. vp, c 3, s 2, sm.
1873. Goodsall, David Henry, F.R.C.S., 17, Devonshire-place, Portland-place, W. vp 2. c, s 2, sm, cfc 7. *Treasurer; Chairman, House and Finance Committee.*
1878. *Gould, Alfred Pearce, M.S., 10, Queen Anne-street, W. vp 2, ll, s 2, c 4, o. *President.*
1896. Gow, William John, M.D., 27, Weymouth-street, W.
1876. Gowers, Sir William Richard, M.D., F.R.S., 50, Queen Anne-street, W. vp, c, sm, ll. fm. 1893.
1896. Grant, J. Dundas, M.D., 18, Cavendish-square, W.
1881. Green, Thomas Henry, M.D., 74, Wimpole-street, W. c 2. vp.
1886. Greves, Edwin Hyla, M.D., Rodney House, Bournemouth.
1873. Grieve, Robert, M.D., C.M.G., Elloughton, Brough, E. Yorks.
1884. Griffiths, Herbert Tyrrell, M.B., 5, Kensington-square, W.
1891. Guthrie, Leonard G., M.D., 15, Upper Berkeley-street, W.

1886. Habershon, S. Herbert, M.D., 88, Harley-street, W. *Councillor.*
1891. Hadley, Wilfred J., M.D., 58, Harley-street, W. c.
1887. Haig, Alexander, M.D., 7, Brook-street, W.
1881. Hall, Charles Ross, M.R.C.S., Hatfield, Herts.
1874. *Hall, Francis de Havilland, M.D., 47, Wimpole-street, W. vp 2, ll, c 7, s 2, sm.
1891. Handfield-Jones, Montagu, M.D., 35, Cavendish-square, W.
1891. Harold, John, M.B., 91, Harley-street, W.
1895. Harrison, Charles James, M.D., Rossetti-mansions, Chelsea, S.W.
1871. Harrison, Reginald, F.R.C.S., 6, Lower Berkeley-street, Portman-square. p, vp 2, ll, c 2.
1883. *Hartridge, Gustavus, F.R.C.S., 12, Wimpole-street, W.
1882. Harvey, John Stephenson Selwyn, M.D., 1, Astwood-road, Cromwell-road, S.W.
1882. Haslam, William Frederick, F.R.C.S., 8, Vicarage-road, Edgbaston, Birmingham. c 3.

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1901. HASLIP, GEORGE ERNEST, M.D., 6, Northumberland-avenue, W.C.
1898. HAYDON, ARTHUR GEORGE, M.D., 17, Essex-road, Horn-lane, Acton, W.
1897. HAYMAN, ALBERT STEPHEN, L.R.C.P., 45, Welbeck-street, W.
1899. HAYS, ALEXANDER GLENCORSE, L.R.C.P., Eydon, Byfield, Northamptonshire.
1890. HEBB, FREDERICK THEODORE, M.R.C.S., 70, Oakley-street, Chelsea-embankment, S.W.
1897. HEDLEY, WILLIAM SNOWDON, M.D., 8, Mansfield-street, W.
1891. HENSMAN, FRANK, M.R.C.S., Lieut.-Colonel, R.A.M.C., Underhill, Sandgate, Kent.
1883. HERMAN, GEORGE ERNEST, M.B., 20, Harley-street, W. C.
1883. HERSCHELL, GEORGE A., M.D., 36, Harley-street, W.
1883. HEWITT, FREDERICK WILLIAM, M.V.O., M.D., 14, Queen Anne-street, W. C.
1892. HILL, WILLIAM, M.D., 26, Weymouth-street, W.
1873. HOBSON, WILLIAM HENRY, M.R.C.S., 38, Leinster-gardens, Hyde Park, W.
1895. HODGSON, GEORGE G. GRAHAM, M.D., The Cedars, Chertsey, Surrey.
1879. HOGG, ARTHUR JOHN, M.R.C.S., Leslie Lodge, Haven-green, Ealing, W.
1884. HOLLAND, CHARLES EDWARD, M.B., "Airdrie," The Avenue, Kew Gardens, Surrey.
1863. HOLMAN, CONSTANTINE, M.D., 26, Gloucester-place, Portman-square, W. VP 2, C 5.
1897. HOLROYDE, JOHN, M.R.C.S., Camden House, Chatham.
1881. HOOD, DONALD WILLIAM CHARLES, C.V.O., M.D., 43, Green-street, W. C 2.
1902. HORNE, MAYNARD, M.B., 37, Nottingham-place, W.
1896. HORSLEY, Sir VICTOR, F.R.C.S., F.R.S., 25, Cavendish-square, W. FM 1896.
1883. *HOVELL, T. MARK, F.R.C.S. Edin., 105, Harley-street, W.
1864. HUME, FREDERICK HENRY, M.D., 53, Devonshire-street, Islington, N.
1889. HUNTER, WILLIAM, M.D., 103, Harley-street, W.
1881. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish-square. P, LL, C 5, O.
1892. HUTCHINSON, JONATHAN, Jun., F.R.C.S., 15, Cavendish-square, W. *Hon. Secretary.*
1875. HUTCHINSON, SAMUEL JOHN, M.R.C.S., 64, Brook-street, W.
1897. HUTCHISON, ROBERT, M.D., 22, Queen Anne-street, W.

1889. I'ANSON, WILLIAM ANDREW, M.R.C.S., Denton Hall, Newcastle-on-Tyne.
1891. ISAAC, G. WASHINGTON, M.B., 75, Gower-street, W.C. C 2.

1884. *JACKSON, FREDERICK WILLIAM, M.D., Yorkgate House, Broadstairs.

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1863. JACKSON, JOHN HUGHLINGS, M.D., F.R.S., 3, Manchester-square, W. P, VP, O, C 5.
1899. JACOBS, HENRY RALPH, M.R.C.S., Culworth, Banbury, Oxon.
1898. JAFFREY, FRANCIS, F.R.C.S., 33, Nottingham-place, W.
1884. *JENNINGS, CHARLES EGERTON, M.D., Burke House, Beaconsfield, Bucks.
1886. JERVIS, ARTHUR, M.D., Pailton Hall, Rugby.
1883. JESSOP, WALTER HAMILTON, F.R.C.S., 73, Harley-street, W. C. *Councillor.*
1886. JOHNSTON, JAMES, M.D., 53, Prince's-square, Bayswater, W.
1893. JOLL, BOYD BURNETT, M.B., 8, Hanger-lane, Ealing, W.
1899. JONES, E. J. TREVOR, M.D., Ty-mawr, Aberdare, S. Wales.
1893. JONES, HENRY LEWIS, M.D., 143, Harley-street, W.
1890. JONES, H. MACNAUGHTON, M.D., 131, Harley-street, Cavendish-square, W.
1902. JONES, JAMES THORESBY, M.D., 103, Sutherland-avenue, Maida-vale, W.
1888. JONES, JOHN TALFOURD, M.B., St. David's, 1, Lascelles-terrace, Eastbourne, Sussex.
1892. *JONES, ROBERT, F.R.C.S., 11, Nelson-street, Liverpool.
1896. JONES, ROBERT, M.D., Claybury Asylum, Woodford Bridge, Essex.
1877. JULER, HENRY EDWARD, F.R.C.S., 23, Cavendish-square, W. C 2.

1889. KAUFFMANN, OTTO JACKSON, M.D., 22, Broad-street, Birmingham.
1891. KEEGAN, DENIS FRANCIS, M.D., Brigade Surgeon-Lieutenant-Colonel, East India United Service Club, St. James's-square, S.W.
1896. KEEP, A. CORRIE, M.C., 14, Gloucester-place, Portman-square, W.
1884. KEETLEY, CHARLES BELL, F.R.C.S., 56, Grosvenor-street, W. C 3.
1899. KELLOCK, THOMAS HERBERT, F.R.C.S., 8, Queen Anne-street, W.
1847. *KELLOCK, WILLIAM BERRY, M.D., 94, Stamford-hill, N.
1891. KELSON, WILLIAM HENRY, M.D., 16, Old Burlington-street, W.
1901. KELYNACK, T. N., M.D., 53, Harley-street, W.
1890. KER, HUGH RICHARD, F.R.C.S. Edin., Tintern, 2, Balham-hill, S.W.
1895. KING, ARTHUR, M.B., Belmont, Station-road, Watford, Herts.
1897. KINGSCOTE, ERNEST, M.B., 31, Lower Seymour-street, W.
1883. KNAPTON, GEORGE, L.R.C.P. Edin., Craven House, Moss-lane, Manchester.
1902. KOENIG, RENÉ PAUL, M.D. (Berne), 42, Welbeck-street, W.

1898. LACK, H. LAMBERT, M.D., 48, Harley-street, W.
1889. LAKE, RICHARD, F.R.C.S., 19, Harley-street, W.
1895. LANE, JAMES ERNEST, F.R.C.S., 46, Queen Anne-street, W.
1881. LANGTON, JOHN, F.R.C.S., 62, Harley-street, W. C 2.
1890. LAW, EDWARD, M.D. Edin., 8, Wimpole-street, W.
1858. *LAWSON, GEORGE, F.R.C.S., 12, Harley-street, W. VP 2, C 3.
1902. LEGG, THOMAS PERCY, F.R.C.S., 141, Harley-street, W.
1887. LEGGATT, CHARLES ASHLEY SCOTT, M.D., 2, Walton-place, S.W.
1894. LENDON, EDWIN HARDING, M.D., 162, Holland Park-avenue, W.
1886. LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., 72, Harley-street, W.

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1902. LEWIS, ERNEST EDWARD, M.D., 30, Weymouth-street, W.
1895. LEWIS, ERNEST WOOL, M.R.C.S., 12, King-street, Hammersmith, W.
1898. LING, MAURICE EDWARD, M.D., 5, West Halkin-street, S.W.
1901. LININGTON, WILLIAM WEST, F.R.C.S., 1, Radnor Park-avenue, Folkestone.
1878. LISTER, the Right Hon. Lord, P.C., O.M., D.C.L., LL.D., F.R.C.S., F.R.S., 12, Park-crescent, Portland-place, W. o.
1896. LISTER, THOMAS D., M.D., 50, Brook-street, W.
1889. LITTLE, ERNEST MUIRHEAD, F.R.C.S., 40, Seymour-street, Portman-square, W.
1889. LITTLE, FLETCHER, M.D., 32, Harley-street, W.
1878. LOCKWOOD, CHARLES BARRETT, F.R.C.S., 19, Upper Berkeley-street, W. VP 2, C 2, S 2.
1881. LORIMER, G., M.D., 9, Terrace-road, Buxton, Derbyshire.
1899. LOW, RICHARD MARSDEN PILKINGTON, M.B., 70, Philbeach-gardens, S.W.
1899. LOW, VINCENT WARREN, F.R.C.S., 27, Queen Anne-street, W.
1897. LUFF, ARTHUR PEARSON, M.D., 9, Queen Anne-street, W.
1889. LUSH, PERCY, M.B., 4, Maresfield-gardens, Hampstead, N.W.
1899. LYNES, JOHN, M.R.C.S., 2, Argyll-road, Kensington, W.

1884. MACBRYAN, HENRY CRAWFORD, L.R.C.P. Edin., Kingsdown House, Box, Wilts.
1894. MCCANN, FREDERICK JOHN, M.B., 5, Curzon-street, Mayfair, W.
1902. MCCLURE, HENRY, M.D., 36, Weymouth-street, W.
1885. MCCONNEL, HENRY WILSON, M.B., Great Ryburgh, Fakenham, Norfolk.
1900. MCENERY, WILLIAM AUGUSTINE, M.R.C.S., Redstone House, Sherborne, Dorset.
1885. MACGEAGH, THOMAS EDWIN FOSTER, M.D., 23, New Cavendish-street, W.
1896. MACGREGOR, PATRICK FRASER, M.D., "Ashley," Cambridge Park, Wanstead, Essex.
1873. MCHARDY, MALCOLM MACDONALD, F.R.C.S. Edin., 5, Savile-row, W.
1894. MACKENZIE, HECTOR WILLIAM GAVIN, M.D., 34, Upper Brook-street, W. C
1880. MACKENZIE, STEPHEN, M.D., 18, Cavendish-square, W. VP 2, C 4, LL, O.
1881. MACLAGAN, THOMAS JOHN, M.D., 9, Cadogan-place, S.W. C 3.
1861. *MACLAREN, ALEXANDER CONNELL, 60, Harley-street, W.
1891. MACLEAN, ALLAN, L.S.A., 71, Peckham-rye, S.E.
1901. MACLEOD, C. E. ALEXANDER, F.R.C.S., 76, Ladbroke-grove, W.
1900. MACLEOD, HERBERT WILLIAM GEORGE, M.D., "The Turret," 28, High-road, Streatham, S.W.
1887. MACREADY, JONATHAN FOSTER CHRISTIAN HORACE, F.R.C.S., 42, Devonshire-street, W. C 2.
1883. MADDICK, EDMUND DISTIN, F.R.C.S. Edin.

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1885. MAGUIRE, ROBERT, M.D., 4, Seymour-street, W. c 5, s 2.
1898. MAHOMED, ARTHUR GEORGE SULIEMAN, M.R.C.S., Astolat, Poole-road, Bournemouth.
1890. MALCOLM, JOHN DAVID, F.R.C.S. Edin., 13, Portman-street, Portman-square, W.
1893. MALCOLM, WILLIAM A., M.B., Oak House, 421, Holloway-road, N.
1887. MANTLE, ALFRED, M.D., Savile-place, Halifax.
1888. MAPOTHER, EDWARD DILLON, M.D., 32, Cavendish-square, W. c 3.
1898. MARRIOTT, CHARLES WILLIAM, M.D., Aubrey House, Bath-road. Reading.
1891. MARSH, HOWARD, F.R.C.S., 30, Bruton-street, W. *Councillor.*
1869. MARSHALL, WILLIAM, M.D., Torrioburn, Barnes, S.W. c.
1889. MARTIN, JOHN MICHAEL HARDING, M.D., Arnheim, Blackburn, Lancashire.
1890. MARTIN, SIDNEY, M.D., F.R.S., 10, Mansfield-street, W. L 3, c.
1893. MASON, GEORGE ARMSTRONG, M.B., 45, George-street, Portman-square, W.
1891. MAUDE, ARTHUR, M.R.C.S., Winterton House, Westerham, Kent.
1892. MAY, CHICHESTER GOULD, M.D., 59, Cadogan-place, S.W.
1891. MAY, WILLIAM PAGE, M.D., 49, Welbeck-street, W.
1884. MEREDITH, WILLIAM APPLETON, F.R.C.S., 21, Manchester-square, W. c.
1897. MILES, WILLIAM ERNEST, F.R.C.S., 17, Devonshire-place, Portland-place, W.
1901. MILLS, HERBERT HENRY, M.B., St. Mary Abbots-terrace, Kensington, W.
1895. MONIER-WILLIAMS, MONTAGU SNEADE FAITHFULL, M.R.C.S., 54, Onslow-gardens, S.W.
1883. MORGAN, JOHN HAMMOND, C.V.O., F.R.C.S., 68, Grosvenor-street, W. P, VP 2, LL, S 2, C 3.
1899. MORIER, C. G. DRUMMOND, L.R.C.P.I., 1, Hamilton-terrace, N.W.
1893. MORISON, ALEXANDER, M.D., 14, Upper Berkeley-street, W.
1895. MORLEY, FRANK, M.R.C.S., 42, Albemarle-street, W.
1881. MORRIS, HENRY, F.R.C.S., 8, Cavendish-square, W. c. *Vice-President.*
1878. MORRIS, MALCOLM ALEXANDER, F.R.C.S. Edin., 8, Harley-street, W. c 2.
1882. MORTON, ANDREW STANFORD, F.R.C.S., 133, Harley-street, W.
1884. *MOULLIN, CHARLES WILLIAM MANSELL, F.R.C.S., 69, Wimpole-street, W. c 2. *Councillor.*
1899. MULLICK, SARAT KUMAR, M.B., 27, New Cavendish-street, W.
1894. MURRAY, CHARLES STORMONT, L.R.C.S. Edin., 85, Gloucester-place, W.
1884. MURRAY, FRED., M.B., Durbanville, Cape Colony, South Africa.
1890. MURRAY, GEORGE, M.R.C.S., 34, Wimpole-street, Cavendish-square, W.
1886. MURRAY, HUBERT MONTAGUE, M.D., 25, Manchester-square, W. c 3.
1879. MURRELL, WILLIAM, M.D., 17, Welbeck-street, W.

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1894. NAUMANN, J. C. FRANCIS, M.D., 125, Gower-street, W.C.
1889. *NIAS, J. BALDWIN, M.D., 5, Rosary-gardens, South Kensington, S.W. c 3.
1880. NIX, EDWARD JAMES, M.D., 11, Weymouth-street, W. c 3.

1887. OAKLEY, ADAM ROBERT HAMILTON, L.R.C.P.
1898. O'BRIEN, CHRISTOPHER, M.D., 57, Amiens-street, Dublin.
1894. O'CALLAGHAN, ROBERT, F.R.C.S.I., 137, Harley-street, W.
1884. OGLE, CHARLES JOHN, M.R.C.S., 1, Cavendish-place, W.
1884. OLIVER, GEORGE, M.D., West End-park, Harrogate.
1892. OPENSHAW, THOMAS HORROCKS, F.R.C.S., 16, Wimpole-street, W.
1899. ORD, GEORGE WILLIAM, M.R.C.S., 58, Queen's-road, Richmond, Surrey.
1892. ORD, WILLIAM WALLIS, M.D., The Hall, Salisbury.
1887. ORMEROD, JOSEPH ARDERNE, M.D., 25, Upper Wimpole-street, W. c.
1889. ORTON, GEORGE HUNT, M.B., 1A, Campden Hill-road, Kensington, W.
1894. OSBORN, SAMUEL, F.R.C.S., J.P., 1A, Devonshire-street, Portland-place, W.
1880. OSWALD, JAMES WADDELL JEFFREYS, M.D., 245, Kennington-road, S.E. c 3.
1901. OWEN, The Rev. A. DUNLEY, M.R.C.S., Russpi, Rhodesia, South Africa.
1883. OWEN, CHARLES J. RAYLEY, M.R.C.S., 14, Devonshire-terrace, Hyde Park, W. c.
1878. *OWEN, EDMUND, F.R.C.S., 64, Great Cumberland-place, W. P, O, VP 2, C 3, S 2, SM, LL. *Trustee.*
1881. OWEN, Sir ISAMBARD, M.D., 40, Curzon-street, Mayfair, W. VP 2, S 2, C 4. *Councillor.*

1880. PALMER, FREDERICK STEPHEN, M.D., 7, Gordon-place, Gordon-square, W.C. c 2. *Vice-President.*
1882. PALMER, WILLIAM PITT, M.B., Kirkham, Babbacombe, Torquay.
1877. *PARAMORE, RICHARD, M.D., 2, Gordon-square, W.C.
1881. PARROTT, EDWARD JOHN, M.R.C.S., The Thorn, Hayes, Middlesex.
1871. PARSONS, FRANCIS HENRY, M.D., "The Hurst," West Worthing.
1885. PASTEUR, WILLIAM, M.D., 4, Chandos-street, Cavendish-square, W. S 2, C. 2. *Trustee.*
1891. PATTERSON, CHARLES SUMNER, M.B., 47, Clarendon-road, Holland-park, W.
1890. PATTISON, EDWARD SETON, M.R.C.S., Granville House, Fulham-road, S.W.
1854. *PAVY, FREDERICK WILLIAM, M.D., F.R.S., 35, Grosvenor-street, W. VP, LL, C.
1881. *PEACEY, WILLIAM, M.D., Rydal Mount, St. John's-road, Eastbourne.
1883. PECK, EDWARD GEORGE, M.A., The Willows, Queensbury, Bradford, Yorks.
1871. PEDLER, GEORGE HENRY, M.R.C.S., 6, Trevor-terrace, Knightsbridge, S.W.

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1883. Perigal, Arthur, M.D., New Barnet, Herts.
1878. Philipps, Sutherland Rees, M.D., 2, Berkeley-place, Cheltenham.
1894. Phillipps, William Alfred, M.D., 13, John-street, Berkeley-square, W.
1876. Phillips, Charles Douglas Ferguson, M.D., F.R.S.E., 10, Henrietta-street, Cavendish-square, W. c 3.
1873. Phillips, George Richard Turner, M.R.C.S., 24, Palace-court, Bayswater-hill, W. c 2.
1883. Phillips, Sidney Philip, M.D., 62, Upper Berkeley-street, Portman-square, W. c.
1883. Pitts, Bernard, F.R.C.S., 109, Harley-street, Cavendish-square. c 5, s 2.
1899. Poore, George Vivian, M.D., 24a, Portland-place, W.
1890. Pope, Harry Campbell, M.D., 280, Goldhawk-road, Shepherd's Bush, W. c 2. *Councillor.*
1902. Potter, Bernard E., M.B., 16, Little Grosvenor-street, W.
1871. *Powell, Sir Richard Douglas, Bart., K.C.V.O., M.D., 62, Wimpole-street, W. p, vp, c 5, o.
1891. Powell, William Wyndham, F.R.C.S., 16, Old Burlington-street, W.
1891. Preston, Theodore Julian, M.R.C.S., Fleet Surgeon, Royal Navy. 238, Portsdown-road, Maida Vale, W.
1891. *Prickett, Marmaduke, M.D., 27, Oxford-square, W.
1885. Pringle, John James, M.B., 23, Lower Seymour-street, W.
1889. Pritchard, Owen, M.D., 41, Gloucester-square, W.
1898. Probyn-Williams, Robert James, M.D., 13, Welbeck-street, W.

1892. Ramsay, James, M.D., High Peter-gate, York.
1894. Rankin, Guthrie, M.D., 4, Chesham-street, Belgravia, S.W.
1881. Ranking, John Ebenezer, M.D., Hanover House, Tunbridge Wells.
1894. Rayner, Herbert Edward, F.R.C.S., Harcourt House, Camberley.
1859. *Rayner, John, M.D., Swaledale House, Highbury-quadrant, N.
1890. Reid, John, M.B., Albert-road, Buckhurst-hill, Essex.
1882. Reid, Thomas Whitehead, M.D., 34, St. George's-place, Canterbury.
1872. *Richards, Joseph Peeke, M.R.C.S., 6, Freeland-road, Ealing, W. c 3.
1899. Richardson, Sidney William F., F.R.C.S., Somerset Hospital, Cape Town.
1868. *Roberts, David Lloyd, M.D., F.R.S.E., 11, St. John's-street, Manchester.
1857. *Roberts, David Watkin, M.D., 56, Manchester-street, W.
1885. Roberts, Edward Coleridge, M.R.C.S., Southgate, N.
1874. Roberts, Frederick Thomas, M.D., 102, Harley-street, W. p, vp 2, o, ll, c 2.
1884. Robinson, Arthur Henry, M.D., St. Mary's Infirmary, Highgate-hill, N. *Councillor.*
1897. Robinson, Henry Betham, M.S., 1, Upper Wimpole-street, W.
1896. Robson, A. Mayo, F.R.C.S., 8, Park-crescent, Portland-place, W., and 7, Park-square, Leeds.

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1902. ROCHE, ALFRED REGINALD, M.R.C.S., 99, Queen's-crescent, Haverstock-hill, N.W.

1897. ROLLESTON, HUMPHRY DAVY, M.D., 55, Upper Brook-street, W. c 3. *Hon. Secretary for Foreign Correspondence.*

1890. ROOT, ARTHUR GUERNSEY, M.D., 46, Eagle-street, Albany, New York, U.S.A.

1886. ROSE, ROBERT DUNCAN, F.R.C.S., 8, St. Leonards, York.

1874. ROSE, WILLIAM, F.R.C.S., 17, Harley-street. c 2, LL, VP 2.

1883. *ROSS, DANIEL MCCLURE, M.D., Cedar Lodge, Littledown-road, Bournemouth.

1888. *ROTH, BERNARD, F.R.C.S., J.P., 38, Harley-street, W.

1893. ROUGHTON, WALTER, F.R.C.S., Cranborne House, New Barnet, Herts.

1881. *ROUTH, AMAND, M.D., 14A, Manchester-square, W. c 5, s 2.

1848. *ROUTH, CHARLES HENRY FELIX, M.D., 52, Montagu-square, W. P, VP 2, O, LL, S 4, C 6, SM. *Trustee.*

1891. RUFFER, MARC ARMAND, M.D., Quarantine Board, Alexandria, Egypt.

1887. RUSHWORTH, FRANK, M.D., "Langdale," Goldhurst-terrace, South Hampstead, N.W.

1889. RUSSELL, JAMES SAMUEL RISIEN, M.D., 44, Wimpole-street, W. *Councillor.*

1886. RUTHERFOORD, HENRY TROTTER, M.D., Salisbury House, Taunton.

1887. SAINSBURY, HARRINGTON, M.D., 52, Wimpole-street, W.

1863. *SANSOM, ARTHUR ERNEST, M.D., 84, Harley-street, W. P, VP, S 2, C 6, SM, §, LL, O.

1895. SANSOM, CHARLES LANE, F.R.C.S., Edin., Public Health Department, Winchester House, Johannesburg (P.O., Box 5382).

1897. SANSOM, HARRY ARTHUR, M.D., The Glen, West End-lane, West Hampstead, N.W.

1886. SAVAGE, GEORGE HENRY, M.D., 3, Henrietta-street, W. c.

1886. SAVILL, THOMAS DIXON, M.D., 60, Upper Berkeley-street, W.

1899. SAYRES, ALEXANDER WARD FORTESCUE, M.D., Lansdown House, Solway-hill, Woodford Green, N.E.

1899. SCHORSTEIN, GUSTAVE, M.B., 11, Portland-place, W.

1873. SEDGWICK, JAMES, M.D., The Firs, Copse-hill, Wimbledon.

1868. SEDGWICK, LEONARD WILLIAM, M.D., 69, Gloucester-terrace, Hyde Park, W. VP 2, C 4, § 3.

1899. SEGUNDO, CHARLES SEMPILL DE, M.B., 6, Brook-street, Hanover-square, W.

1883. SEMON, Sir FELIX, M.D., 39, Wimpole-street, W. §, C.

1887. SERVAIS, LEOPOLD, M.D., Antwerp, Belgium.

1889. SHAW, GEORGE, M.B., 1, The Drive, West Brighton.

1884. SHAW, JOHN, M.D., 32, New Cavendish-street, Cavendish-square, W.

1886. SHEILD, ARTHUR MARMADUKE, F.R.C.S., 4, Cavendish-place, W. c 2, s 2.

1890. SHEPPARD, WILLIAM JOHN, M.D., 211, Upper Richmond-road, Putney, S.W.

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1885. SHOEMAKER, JOHN V., M.D., 1031, Walnut-street, Philadelphia, U.S.A.
1894. SHUTTLEWORTH, GEORGE EDWARD, M.D., Ancaster House, Richmond, Surrey.
1890. SILK, JOHN FREDERICK WILLIAM, M.D., 47, Devonshire-street, W.
1884. SIMPSON, JAMES HERBERT, M.D., Hillmorton-road, Rugby, Warwickshire.
1898. SIMPSON, JOHN POLLOCK, M.D., 1, Blandford-street, Manchester-square, W.
1884. SINCLAIR, JOHN, M.D., General Post Office, St. Martin's-le-Grand, E.C.
1883. *SKERRITT, EDWARD MARKHAM, M.D., Richmond Hill, Clifton.
1886. SLATER, CHARLES, M.B., 81, St. Ermin's-mansions, Westminster, S.W.
1862. *SLIGHT, GEORGE, M.D., 14, Old Burlington-street, W. C 2.
1889. SMALE, MORTON, M.R.C.S., 22A, Cavendish-square, W. C 3.
1845. *SMILES, WILLIAM, M.D., St. Martha's Lodge, Guildford. VP 2, S 4, C 9, SM.
1897. SMITH, A. LIONEL H., M.R.C.S., Addenbrooke's Hospital, Cambridge.
1896. SMITH, E. STANLEY, M.D., 68, Wimpole-street, W.
1887. *SMITH, FREDERICK JOHN, M.D., 138, Harley-street, W. C 2.
1873. SMITH, HEYWOOD, M.D., 25, Welbeck-street, W. C 3.
1880. SMITH, NOBLE, F.R.C.S. Edin., 24, Queen Anne-street, W.
1891. SMITH, SOLOMON CHARLES, M.D., Four Oaks, Walton-on-Thames.
1877. SMITH, SYDNEY LLOYD, M.R.C.S., 25, Argyle-square, King's Cross, W.C.
1882. SMITH, THOMAS FREDERICK HUGH, F.R.C.S., Farningham, Kent.
1873. *SMITH, THOMAS GILBART, M.D., 68, Harley-street, W. VP 2, S 2, SM, C 4. *Trustee.*
1872. SMITH, WALTER, M.R.C.P. Edin., Orleans House, 60, Regent's Park-road, N.W.
1899. SMITHSON, OLIVER, M.R.C.S., Moor-street, Luton, Beds.
1874. SMYTH, WILLIAM WOODS, L.R.C.P. Edin., Maidstone.
1894. SNAPE, ERNEST ALFRED, M.D., 41, Welbeck-street, W.
1893. SPENCER, HERBERT RITCHIE, M.D., 104, Harley-street, W. C.
1901. SPENCER, WALTER GEORGE, M.S., 35, Brook-street, W.
1887. SPICER, SCANES, M.D., 28, Welbeck-street, W.
1883. SPITTA, EDMUND JOHNSON, M.R.C.S., Ivy House, 31, South Side, Clapham Common, S.W.
1864. SQUIRE, ALEXANDER JOHN BALMANNO, M.B., 24, Weymouth-street, Portland-place.
1881. STARTIN, JAMES, M.R.C.S., 15, Harley-street, W.
1892. STAVELEY, WILLIAM H. C., F.R.C.S., 13, South Eaton-place, S.W.
1896. STEELE-PERKINS, GEORGE CHAPMAN, M.D., 85, Wimpole-street, W.
1884. STEPHENS, WILLIAM JOHN, L.R.C.P., 25, Clermont-terrace, Preston, Brighton.
1901. STEWART, HUGH MILTON, M.D., Walton House, Lordship-lane, S.E.
1882. STEWART, JAMES, F.R.C.P. Edin., Dunmurry, Sneyd Park, near Clifton.
1894. STEWART, KENNETH TREVOR, M.D.

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1883. Stewart, William Robert Henry, F.R.C.S. Edin., 42, Devonshire-street, Portland-place, W.
1885. Stivens, B. H. Lyne, M.D., 107, Park-street, Grosvenor-square, W.
1848. *Stocker, John Sherwood, M.D. c 10, s 2.
1884. Stoker, George, C.M.G., M.R.C.S., J.P., 14, Hertford-street, Mayfair, W.
1892. Stonham, Charles, C.M.G., F.R.C.S., 4, Harley-street, W.
1877. Stowers, James Herbert, M.D., 128, Harley-street, W.
1873. Strange, William Heath, M.D., 2, Belsize-avenue, Hampstead, N.W. c 3.
1881. Sturge, William Allen, M.D., Maison Malaussena, Boulevard Dubouchage 29, Nice. sm.
1889. Sumpter, Walter John Ernely, M.R.C.S., Sheringham, Norfolk.
1892. Sunderland, Septimus, M.D., 11, Cavendish-place, W.
1896. Swanton, James Hutchinson, M.D., 40, Harley-street, W.
1892. Swift, William John Cropley, M.R.C.S., 4, Gordon-square, W.C.
1885. *Syers, Henry Walter, M.D., 75, Wimpole-street, W.
1884. Symonds, Horatio Percy, F.R.C.S., 35, Beaumont-street, Oxford.
1893. Symonds, Charters James, F.R.C.S., 58, Portland-place, W. c.
1899. *Sympson, Edward Mansel, M.D., Deloraine Court, Lincoln.

1864. Tait, Edward Wilmshurst, M.R.C.S., 10, Ellerdale-road, Hampstead, N.W.
1882. Taylor, Seymour, M.D., 16, Seymour-street, Portman-square, W. c 2.
1897. Templeton, George, F.R.C.S., 10, Seymour-street, Portman-square, W.
1898. Thomas, John Lynn, C.B., F.R.C.S., 28, Charles-street, Cardiff.
1859. *Thompson, Edmund Symes, M.D., 33, Cavendish-square, W. vp, o, s 3, c 3, sm.
1855. *Thompson, Sir Henry, Bart., F.R.C.S., 35, Wimpole-street, W. vp, ll, c 4.
1900. Thompson, Henry E. Symes, M.R.C.S., 33, Cavendish-square, W.
1896. Thompson, Henry George, M.D., 86, Lower Addiscombe-road, Croydon, Surrey.
1895. Thompson, James Andrew Baird, M.D., White Hall, Abridge, Essex.
1873. Thomson, John Roberts, M.D., Monkchester, Bournemouth, Hants.
1894. Thomson, StClair, M.D., 28, Queen Anne-street, W.
1898. Thorne, Atwood, M.B., 10, Nottingham-place, W.
1899. Thorne, W. Bezly, M.D., 53, Upper Brook-street, W.
1897. Thornton, Bertram, M.R.C.S., J.P., Berkeley Lodge, Trinity-square, Margate.
1892. Thornton, George, M.D., Pretoria Hospital, Pretoria, South Africa.
1876. Thornton, John Knowsley, M.C., J.P., Hildersham Hall, Cambs. p, vp, c 3.
1884. Thursfield, Thomas William, M.D., J.P., Selwood, Beauchamp-square, Leamington.
1865. Travers, William, M.D., 2, Phillimore-gardens, Kensington, W.
1897. Tresilian, Frederick James, M.D., Redlingtons, Enfield.

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1884. *Treves, Sir Frederick, Bart., C.B., K.C.V.O., F.R.C.S., 6, Wimpole-street, W. c 3, ll, vp 2.
1899. Tubby, Alfred Herbert, M.S., 25, Weymouth-street, W.
1882. Tuke, Charles Molesworth, M.R.C.S., Chiswick House, Chiswick.
1886. Tuke, Thomas Seymour, M.B., Chiswick House, Chiswick.
1884. Turner, George R., F.R.C.S., 41, Halfmoon-street, Piccadilly, W. vp 2, s 2, c 5.
1890. Tweed, Edward Reginald, M.D., Hembury Fort, Honiton, Devon.
1883. Tweedy, John, F.R.C.S., 100, Harley-street, W. c.
1891. Tyson, William Joseph, M.D., 10, Langhorne-gardens, Folkestone.

1887. *Underwood, Edward T., M.D., Fort Bombay, India.

1897. Vassie, Alexander Henry, M.B., 98, Priory-road, West Hampstead, N.W.
1883. Venning, Edgcombe, F.R.C.S., 30, Cadogan-place, S.W. *Councillor.*
1899. Vise, Christopher, M.D., 39, Mount Pleasant-road, Tunbridge Wells.
1893. Voelcker, Arthur Francis, M.D., 101, Harley-street, W. s 2, c. *Hon. Librarian.*

1892. Waggett, Ernest Blechynden, M.B., 45, Upper Brook-street, W.
1850. *Waggett, John, M.D., Perivale, Bournemouth.
1884. Wakley, Thomas, Jun., L.R.C.P. Lond., 5, Queen's-gate, S.W.
1850. *Wakley, Thomas Henry, F.R.C.S., 5, Queen's-gate, S.W.
1898. Wallace, Cuthbert Sidney, F.R.C.S., St. Thomas's Hospital, S.E.
1894. Wallis, Frederick Charles, F.R.C.S., 107, Harley-street, W. s 2, c 2.
1901. Walsham, Hugh, M.D., 114, Harley-street, W.
1880. Walsham, William Johnson, F.R.C.S., 77, Harley-street, W. c.
1895. Walters, Frederick R., M.D., Crooksbury Sanatorium, Farnham, Surrey.
1899. Ward-Humphreys, George Herbert, M.R.C.S., 9, Cavendish-place, W.
1894. Waring, Holbert Jacob, F.R.C.S., 37, Wimpole-street, W.
1894. Waterhouse, Herbert Furnivall, F.R.C.S., 81, Wimpole-street, W. c 2. *Councillor.*
1883. Waterhouse, William Dakin, LL.D., 50, West End-lane, West Hampstead, N.W. c.
1889. Waugh, Henry Dunn, M.D., 6, Sumner-place, Onslow-square, S.W.
1884. Webb, F. Ernest, M.R.C.S., 113, Maida-vale, W.
1896. Weber, Frederick Parkes, M.D., 19, Harley-street, W.
1889. Weber, Sir Hermann, M.D., 10, Grosvenor-street, W.
1901. Weekes, Henry Holman, M.D., 21, Kidbrook-park-road, Blackheath, S.E.
1898. Weeks, Courtenay Charles, M.R.C.S., 9, Lewisham-park, S.E.
1838. *Wells, John Robinson, F.R.C.S., 4, Pierrepoint-road, Acton, W. c 2.

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1884. West, Samuel, M.D., 15, Wimpole-street, W. vp 2, s 2, c 5, cfc, ll.
1889. Wethered, Frank Joseph, M.D., 83, Harley-street, W.
1892. Wheaton, Samuel Walton, M.D., 10, Rastell-avenue, Streatham-hill, S.W.
1882. Whipham, Thomas T., M.D., 11, Grosvenor-street, W. sm, c. *Vice-President.*
1899. White, Charles Percival, M.B., 22, Cadogan-gardens, S.W.
1889. White, Edwin F., F.R.C.S., Westlands, 280, Upper Richmond-road, Putney, S.W. c. *Councillor.*
1880. *White, William Henry, M.D., 43, Weymouth-street, W. c. 3.
1883. Whitehead, Walter, F.R.C.S. Edin., F.R.S.E., 499, Oxford-road, Manchester. c.
1885. Whitla, Sir William, M.D., 8, College-square North, Belfast, Ireland.
1898. Willey, F. Ingor, M.B., The Wych, 8, Avenue-road, Highgate, N.
1872. Williams, Charles Theodore, M.D., 2, Upper Brook-street, Grosvenor-square, W. p, vp 2, ll, s 2, sm, o, l 3, c 9.
1876. Williams, Henry William, M.D., Hillside, Guilsborough, Northamptonshire.
1883. Williams, Sir John, Bart., M.D., 63, Brook-street, W. c 3.
1900. Willock, Edward Hulse, M.R.C.S., 113, London-road, Croydon.
1873. Wills, Thomas Munns, F.R.C.S.I., J.P., 44, Merton-road, Bootle, Liverpool.
1892. Wilson, Claude, M.D., Belmont, Tunbridge Wells.
1901. Wilson, John Clark, M.D., 22, Hilgrove-road, South Hampstead, N.W.
1884. Winslow, H. Forbes, M.D., 14, York-place, Portman-square, W.
1873. Winslow, Lyttleton Stewart Forbes, M.B., D.C.L., LL.D., 33, Devonshire-street, W. c.
1876. Woakes, Edward, M.D., 78, Harley-street, W.
1900. Wood, Guy M., M.B., 6, Woburn-square, W.C.
1886. Wood, T. Outterson, M.D., 40, Margaret-street, Cavendish-square, W. c 3.
1873. Woodhouse, Robert Hall, M.R.C.S., 1, Hanover-square, W.
1891. Woollett, Charles Jerome, F.R.C.S., "Ambleside," Streatham, S.W.
1902. Wrangham, William, M.D., Cranborne-corner, Ascot.
1898. Wright, Sydney Faulconer, M.D., St. Peter's Lodge, Eltham-road, Lee, S.E.
1902. Wynter, W. Essex, M.D., 30, Upper Berkeley-street, W.

1891. Yarr, Michael Thomas, Major, R.A.M.C., St. Ives, Farnborough-road, South Farnborough, Hants.
1884. Yeo, I. Burney, M.D., 44, Hertford-street, Mayfair, W.
1902. Young, Robert Arthur, M.D., 41, Wimpole-street, W.
1897. Young, Williams H. Frome, M.R.C.S., Malcolm Corner, Worple-road, Wimbledon.
1884. Younger, Edward George, M.D., 19, Mecklenburgh-square, W.C. c 3.

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NON-SUBSCRIBING FELLOWS.

1868. BATEMAN, Sir FREDERIC, M.D., J.P., Upper-street, Giles-street, Norwich.
1872. BELL, JOHN HOUGHAM, M.D., Ventnor, Isle of Wight.
1868. BUCKLE, FLEETWOOD, M.D., Staff Surgeon, R.N., Merton Lodge, Southsea.
1870. CLOUSTON, THOMAS SMITH, M.D., Royal Asylum, Morningside, Edinburgh. FM 1870.
1868. FOLKER, WILLIAM HENRY, F.R.C.S., Hanley, Staffordshire.
1869. FOSTER, Sir WALTER B., M.D., M.P., 14, Temple-row, Birmingham.
1868. FOX, CHARLES HENRY, M.D., 35, Heriot-row, Edinburgh.
1868. GAINE, CHARLES, Weston Lea, near Bath.
1871. GLYNN, THOMAS ROBINSON, M.D., 62, Rodney-street, Liverpool.
1868. KNAGGS, SAMUEL, 2, Bradley-lane, Huddersfield.
1869. LEES, CHARLES ALEXANDER, M.D., Dep. Inspector-General, R.N.
1869. MATHEWS, ROBERT, Bickley, Kent.
1871. MAURICE, OLIVER CALLEY, M.D., J.P., 75, London-street, Reading.
1868. McINTYRE, JOHN, M.D., LL.D., Odiham, Hants.
1868. NEVINS, JOHN BIRKBECK, M.D., 32, Princes-avenue, Liverpool.
1871. OGLE, WILLIAM, M.D., The Elms, Derby.
1869. PHILIPSON, Sir GEORGE HARE, D.C.L., M.D., J.P., 7, Eldon-square, Newcastle-on-Tyne.
1869. PRICE, WILLIAM PRESTON, M.D., 1, Ethelbert-crescent, Margate.
1869. PRIOR, CHARLES EDWARD, M.D., 4, Goldington-road, Bedford.
1869. ROBERTS, BRANSBY, M.D., Raven's Moat, Carlisle-road, Eastbourne.
1869. STEAR, HENRY, Saffron Walden, Essex.
1869. TAYLOR, CHARLES BELL, M.D., 9, Park-row, Nottingham.
1869. WALKER, JOHN SWIFT, M.D., Hanley, Staffs.

*** As it is very desirable that the foregoing Lists should be kept as accurately as possible, Fellows are requested to send notice of any corrections that may be necessary to the Secretaries or to the Registrar.

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GENERAL MEETING.

May 12th, 1902.

WILLIAM HENRY ALLCHIN, M.D., F.R.C.P., *President*, in the Chair.

Report of the Council for the Session 1901–1902.

THE Council beg to present their Report for the Session 1901–1902.

The total number of Fellows is now 718, being 5 more than last year. There is a satisfactory increase in the number of new Fellows elected, 28 having joined this Session. The details for the last three years are given in the following table :—

—	1899–1900.			1900–1901.			1901–1902.		
	Ord.	Hon., &c.	Total.	Ord.	Hon., &c.	Total.	Ord.	Hon., &c.	Total.
At beginning of Session	662	85	747	650	79	729	640	73	713
Elected in Session ...	21	1	22	15	1	16	28	—	28
	683	86	769	665	80	745	668	73	741
Less—									
Deaths	10	7	17	14	7	21	3	4	7
Resignations	14	—	14	10	—	10	12	—	12
Struck off	9	—	9	—	—	—	4	—	4
Made Hon. Fellow ...	—	—	—	1	—	1	—	—	—
Total Decrease ...	33	7	40	25	7	32	19	4	23
Total at end of Session ...	650	79	729	640	73	713	649	69	718
Nett Increase or Decrease	−12	−6	−18	−10	−6	−16	+9	−4	+5

The following deaths were reported during the Session :—Honorary Fellows—Sir William MacCormac, Bart. (President, 1888), and M. Le Roi de Mericourt ; Corresponding Fellows—Mr. A. Beardsley and Mr. T. G. Stockwell ; Other Fellows—Mr. T. Bond, Dr. J. L. W. Thudichum, and Dr. H. Sutherland.

The Annual Oration, which was delivered by Mr. Richardson Cross, on "Some Landmarks in the Progress of Medical Science," was well attended.

The Lettsomian Lecturer, Mr. A. Pearce Gould, took as his subject "Certain Diseases of the Blood-Vessels." The Lectures were full of

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valuable and suggestive observations on a subject of much practical importance, and formed an interesting complement to the Lettsomian Lectures of the last Session.

The Fothergillian Gold Medal for 1902 was awarded to Dr. Patrick Manson, C.M.G., F.R.S., "in recognition of the value of his work on Malaria and Tropical Diseases."

The Anniversary Dinner was a great success, and was attended by 130 Fellows and their friends.

During the past Session the attendance at the Meetings has been above the average. Three set debates—one each on a Medical, Surgical, and Gynæcological subject—have been held, and have been eminently successful.

The new arrangements made for the conduct of the Clinical Evenings have proved quite satisfactory.

Report of the House and Finance Committee for the Session 1901–1902.

The Committee have the pleasure to report that the financial position of the Society continues to be satisfactory. The receipts from rents and occasional lettings remain practically the same as last year.

One Debenture Bond has been redeemed, making a total of £1,200 paid off within the last six years, and leaving the sum of £2,400 still outstanding.

The Meeting-room, Library, and Entrance Hall require to be cleaned and repainted. It is proposed to have the work done before the commencement of the next Session.

(Signed) D. H. Goodsall,
Chairman.

May 9th, 1902.

Report of the Honorary Librarian, Session 1901–1902.

I have the honour to report that the condition of the Library for the year ending April 30th, 1902, has been satisfactory. The number of volumes borrowed from the Library has been 173, while from Lewis's Library 71 volumes have been borrowed. Since January 1st, 1902, the Society's subscription to Lewis's Library has been increased from seven guineas (20 volumes) to 10 guineas (30 volumes), thus giving an increased facility for Fellows to obtain the most recent books which the Society may be unable to purchase.

The additions to the Library during the year, exclusive of periodicals, were: purchased, 11 volumes; presented by authors and publishers, 15 volumes; second-hand works presented by Fellows, 129 volumes; making a total of 155 volumes. The second-hand works include 15 volumes of the 'Real Encyclopädie der Gesammten Heilkunde,' and 60 volumes of the 'Dictionnaire Encyclopedique des Sciences Medicales,' presented by Dr. Leonard Sedgwick; 38 volumes of 'Canstett's and Virchow's Jahresbericht,' presented by Sir William Gowers; 4 volumes in MS. containing the 'Minutes of the Lyceum Medicum Londinense,' with six other volumes of early medical works presented by Dr Isambard Owen.

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Donations have also been received from Dr. W. H. Allchin (the President), Mr. C. A. Ballance, Surgeon-Major Black, Dr. Bulkley, Dr. E. H. Lendon, Dr. Macnaughton-Jones, Mr. Henry Morris, Dr. T. H. Pegler, Dr. Steele-Perkins, and Sir Henry Thompson, Bart.

No further rebinding of the earlier works has been carried out during the year.

Dr. J. B. Nias has completed his examination of the Greek MSS., and finds them in good condition, and the majority of considerable intrinsic value. The cataloguing of the MSS. is incomplete, and ought to be attended to.

Appended is a list of the principal new works acquired :—

ALLCHIN (W. H.). 'Manual of Medicine.' Vol. iii.
AULD (A. G.). 'Selected Researches in Pathology.'
BALLANCE (C. A.) and STEWART (P.). 'The Healing of Nerves.'
BULKLEY (L. D.). 'Syphilis in the Innocent.'
CABOT (R. C.). 'Clinical Examination of the Blood.' 3rd edition.
CHEYNE (W.) and BURGHARD (F.). 'Surgical Tretament.' Parts v and vi, Section i.
EWALD (C. A.). 'Klinik der Verdauungskrankheiten.' Bd. iii.
HARE (H. A.). 'Progressive Medicine, 1901.' 4 volumes.
HUTCHISON (R.). 'Food and Dietetics.' 2nd edition.
LENDON (E. H.). 'The Method of Cuignet or Retinoscopy.'
LIMBECK (R. W.). 'Clinical Pathology of the Blood.' (N. Syd. Soc.)
MACNAUGHTON-JONES (H.). 'Diseases of Women and Uterine Therapeutics.' 8th edition.
——————— 'Practical Points in Gynæcology.' 2nd edition.
MORRIS (H.). 'Surgical Diseases of the Kidney and Ureter.' 2 volumes.
PEGLER (L. H.). 'Nasal Insufficiency.'
THOMPSON (Sir HENRY). 'Modern Cremation.' 4th edition.
——————— 'Food and Feeding.' 11th edition.
TREVES (Sir F.). 'Surgical Applied Anatomy.' New edition.

(Signed) SIDNEY MARTIN,
Hon. Librarian.

May 9th, 1902.

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MEDICAL SOCIETY

DR. BALANCE SHEET, FROM 1ST OCTOBER,

RECEIPTS.

			£ s. d.	£ s. d.
Ordinary—				
Subscriptions			502 19 0	
Entrance Fees			26 15 6	
Life Composition Fees			23 2 0	
Rents	Bolt Court	£84 15 0		
	Chandos Street	743 10 6		
	Occasional Lettings ..	57 15 0		
			886 0 6	
Sale of 'Transactions'			1 17 6	
				1,440 14 6
				£1,440 14 6

FOTHERGILLIAN

	£ s. d.
Balance from Last Account	85 11 4
Dividend on £916 10*s.* 5*d.*, 2¾ % Consols	25 4 0
	£110 15 4

Examined, compared with vouchers, and found correct.

(Signed) F. J. McCANN, T. H. KELLOCK, } *Auditors.*

4th October, 1902.

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OF LONDON.

1901, TO 30TH SEPTEMBER, 1902. CR.

EXPENDITURE.

	£	s.	d.	£	s.	d.
Balance outstanding, Last Account	..		..	13	13	6
Ordinary—						
Rent, 11 and 12, Chandos Street	282	10	0			
Rates, Taxes, and Insurance	215	11	5			
Registrar's Salary and Allowances	138	11	0			
Collector's Poundage on Subscriptions	25	2	11			
Reporter's Salary	20	9	6			
Stationery and Printing	18	9	0			
Printing and issuing 'Transactions'	135	5	11			
Library Expenses	22	18	1			
Postage and Stamps	10	11	8			
Coals and Wood	12	5	9			
Gas and Electric Supply	52	1	11			
Repairs	19	8	5			
Interest on Debentures	91	11	11			
Conversazione	45	0	0			
Refreshments at Meetings	14	4	6			
Patients' Expenses, Clinical Meetings	3	8	6			
Lantern and Microscopic Demonstrations ..	2	17	0			
Bankers' Charges, 1*s.* 3*d.*; Travelling, 2*s.*; Wreath, £1 5*s.*	1	8	3			
Wages	46	15	6			
Chandlery and Cleaning	13	9	1			
Anniversary Dinner £79 1 0						
Less Fellows' Tickets 63 0 0						
	16	1	0			
Extraordinary—				1,188	1	4
Redemption of One Debenture				100	0	0
Balance at Bank, 30th September, 1902				138	19	8
				£1,440	14	6

FUND.

	£	s.	d
Books Purchased for Library..	7	2	10
Gold Medal awarded to Dr. P. Manson, C.M.G., F.R.S... ..	21	0	0
Cheque Book	0	5	0
Balance at Bank, 30th September, 1902	82	7	6
	£110	15	4

(Signed) D. H. GOODSALL, *Treasurer.*

2nd October, 1902.

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TRANSACTIONS

OF THE

MEDICAL SOCIETY OF LONDON.

129TH SESSION.

October 14*th*, 1901.

OPENING ADDRESS ON THE RESPONSIBILITY OF THE ORGANISM IN DISEASE.

By the President, WILLIAM HENRY ALLCHIN, M.D., F.R.C.P. Lond.

FELLOWS OF THE MEDICAL SOCIETY AND GENTLEMEN,—Among the widest spread inclinations of the human mind, however uncultivated or however learned, is the desire to ascertain the cause of such phenomena as come under observation. Instinctively one seeks for the precedent condition to which may be attributed the event we are concerned with, and it is scarcely going too far to say that when once this is ascertained further interest in the subject not infrequently lapses. That this is the case in respect to disease, so far as the laity is concerned, is, I take it, well known to most of us, and our patients after the malady is named at once are concerned to inquire its cause, and are generally fully satisfied by ascribing it to "a chill," more or less real, or in finding some ancestor supposed to have similarly suffered. Although to the pathologist and physician such references are but partially convincing, it may be noted that these explanations typify the two main groups of conditions to which may be reduced the varying circumstances that prejudicially affect our well-being, contrasting, as they do, the individual and his inheritance on the one hand, and on the other the external conditions to which he is subjected.

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At the risk of offering you no novelty, and of dealing with a subject that is largely conjectural in its bearings, though not entirely without practical application, I propose to occupy the occasion which pertains to the position you have summoned me to by offering some remarks upon that aspect of the subject which more particularly concerns the individual, and to consider how far he is himself responsible for any given condition of disease. And I may say that I have been somewhat led to this choice of subject by what, unless I am mistaken, appears to be an indication that in the far-reaching and all-important extension of our knowledge, by the discovery of the part played by microbes in the production of morbid states, that the personal factor is running the risk of being somewhat lost sight of, if not admittedly, at least in fact.

The Present Conception of the Nature of Life.

The conception of life as held at the present day, and expressed in terms that are consistent with our general notions of the cosmos, is that it is the sum total of the phenomena exhibited by certain bodies, resulting from the interaction between a special constituent of those bodies—bioplasm (cell)—and the environment. Thus stated there are clearly two factors to be considered, in the modifications of the one or other of which are to be found the immediate and predisposing causes of altered vitality, *i.e.*, disease, whilst the resulting phenomena themselves, independent to some extent of the circumstances of their causation, afford a field of study that furnishes the various signs and symptoms of the different maladies we are called upon to investigate, and that should for any intelligent appreciation of them be compared with the corresponding manifestations that are taken as normal.

With the shifting point of view that increasing knowledge has furnished, now advancing as the knowledge is confirmed, now falling back as some false wind of doctrine has diverted inquiry from its true course, these two aspects of the subject have been variously credited with importance or the reverse, as being responsible for the causation of disease. The most superficial retrospect of the past in this respect will show how opposite are the views that have prevailed. In this, as in many other points of pathological doctrine, we find the earliest recorded Hippocratic teaching strangely similar to that of the present day. There were the several "temperaments"

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corresponding to the four fundamental "humours," a discrepancy in the mixture of which, and an undue preponderance of either would be the cause of disease, which clearly represents the responsibility of the individual; but equally did the Father of Medicine teach that disease might arise from external influences, such as air, season, climate, water, upon which he wrote largely. No such complete conception of the ætiology of disease has existed until recent times, erroneous as, of course, was much of the substructure. One or other factor was from time to time held as the all-important, though it would probably be correct to say that the most generally prevalent notions ascribed to the individual rather than to his surroundings, the determining circumstances of disease, and this notwithstanding those who taught the entrance *ab extra* of evil spirits, which brought about each its peculiar malady. The idea of disease as a correlative of health, and both as aspects of life, did not enter into the conceptions of the ancients. Disease was with them a something superadded to the body, whatever its origin, and the long-maintained doctrines of the humoralists, as also the very different doctrines of the vitalists in their numerous modifications, saw in flaws in the humours or fluids of the body, or in errors of a vital principle contained in the body, an explanation of disease. The dependence of function, whether healthy or morbid, upon structure is of modern conception, and it is left still to us to discriminate how far the departures of the structure from a healthy standard are due to intrinsic and how far to extrinsic conditions.

Although the main purport of my remarks is the share taken by the individual in the causation and manifestation of disease, yet it is necessary to an adequate understanding of my theme that I should say a few words upon the external conditions to which every living being is subject.

The Environment.

In proceeding to consider the surrounding circumstances of the organism, which in some measure and degree are essential to its well-being, but that beyond those normal limits become sources of harm and the causes of so-called acquired disease, it must be remembered that the investigation of their action must not be limited to their effects, more or less obvious, upon the body *en masse*, but must extend further and include if possible the influence which they exert upon the component cells and tissues, and especially upon the

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bioplasm itself; and on this subject a large amount of experimental evidence has been accumulated.*

For present purposes the environmental factors may be thus grouped:—

(i) Violent contact, represented by blows, injuries, and the laceration of internal structures by foreign bodies, such as calculi and the like. As the causes of maladies, arbitrarily termed surgical, since their treatment mainly involves manual skill, they are obvious; but it is not to be forgotten that in infinitely less degree only, is mere contact a normal provocative of those metabolic changes which find expression in contraction, movement, and locomotion. Just as the loosely-associated molecules in those chemical compounds known as explosives are rearranged with great display of force when thrown into vibration, so parts, at least, of the highly complex and unstable substance bioplasm are similarly disturbed, with the manifestation, among other things, of energy termed contractile, nervous, and possibly secretory.

(ii) Another group of external conditions is represented by such natural agents as heat, light, and electricity, and as barometric pressure, gravity, and such subsidiary conditions as the relative humidity or dryness of the atmosphere. With extremes of any one or more we recognise the occurrence of disease in various forms, sometimes with no difficulty, at others only vaguely and with uncertainty, as, for instance, in the different degrees of malaise or positive illness that we associate with change of weather, itself a complex of these various agencies. But, although there is much obscurity in detecting the exact effects that are produced upon the body as a whole by these ever-present conditions, in the innumerable combinations in which they occur, we have very precise knowledge concerning some of them in their separate action upon isolated living cells, and there is as little doubt that they may become responsible for disease as there is for the absolute dependence upon them of life, or the benefit they confer when employed as therapeutic agents.

(iii) A third set of conditions that are brought to bear upon the body and its component parts from outside are those which produce their effects in virtue of the chemical changes which they set up. Therein will be included modifications of the ingesta,—solid, liquid,

* 'Experimental Morphology,' by Dr. Davenport: Macmillan, 1897.

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and gaseous,—and perversions of the alterations they may undergo in the process of digestion or absorption; in fact, the whole class of substances denominated poisons, of whatever source. As causes of disease these bodies are well known, and the recent development of bacteriology and a knowledge of the toxins elaborated by micro-organisms has widely extended our acquaintance with agents of this group, and consequently of our understanding of the ætiology of many maladies, perhaps at the risk of overlooking some of the other, and even more subtle and less understood agencies previously enumerated. Investigations into the mode of action of poisons upon living protoplasm show that they act by altering the minute structural arrangement or chemical constitution in various ways. Thus some determine an oxidation of the proteid, carbohydrate, or fat constituents of the protoplasm, as some act by substituting chemical elements or radicals for others in these components, and the general outcome is that the toxic properties of each substance are closely connected with the complexity and instability of its composition.

Although these several groups of agents are spoken of as external, that term is not to be taken as necessarily implying extrinsic to the entire organism, for in the inconceivably complicated structure of the higher forms of life, cells and groups of cells and tissues undoubtedly exert an influence beneficial or otherwise upon other cell-groups, quite apart from what is occurring outside the body as a whole. Here it is that we should place the autogenetic poisons resulting from perversions of tissue metabolism; and it is at least conceivable that influences, favourable or inimical, are mutually excited among bioplasmic units of a character which we cannot at present formulate, but which are familiar to us in their remote results, such as the effects of internal secretions, or of those peculiar forms of nervous stimuli which we designate trophic. Herein also would be included the consequences of the mechanical pressure exerted by new growths, inflammatory exudations, and the like, upon adjacent tissues, which are manifested as degenerations, necrosis, &c.

In this extended view of the nature of the environments, the influence, that is, which may be exerted by cells and tissues of an organism upon other component cells and tissues of the same, should lead us to realise the exact bearing which the so-called complications and sequelæ hold to the original malady. The perverted

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intonations which ensue upon the infliction of the primary causes in their turn become disturbing factors in the life course of cells that are remote from the original seat of mischief, and lead to the manifestation of morbid symptoms that may altogether overshadow, both in importance and extent, those of the primary disease. Whilst, however, in the strict analysis of the condition the causation of these complications is to be found in the environment of the tissues affected, it is impossible, from the practical point of view, to regard them as other than those for which the individual is responsible, and to which the actual condition of the secondarily involved structures contributes not a little.

If we endeavour to penetrate still more closely into the way in which these several agencies affect the bioplasm, it is needful to remember the extraordinary complexity of this substance, which so eludes precise investigation of its composition, since it is altered and ceases to retain its living characteristics by the very means adopted for its analysis. Enough is, however, known to justify the view that its structure resembles that of a foam consisting of incomplete loculi formed by delicate strands and septa in the meshes of which the serous fluid, or at least diffluent material, is contained. Entering into the composition of this highly complex, profoundly unstable substance are proteid molecules, as well as fat and carbohydrate molecules and salts, the intimate chemical relations of which—the chemical structure, so to say, for this, to my mind, is but the ultimate stage of its anatomy—are quite unknown. Now the action of the environment upon this material essentially consists in bringing about alterations in its structure and constitution, and with that affecting its functional capacity. Extreme conditions, such as great heat or cold, excessive electrical stimulation, &c., so affect its integrity as to determine a state of inaction, which is either suspended vitality or death according as the state be recoverable from or not. Gross molar injuries disturb with violence the complex structure; variations in degree of moisture by the relative amount of desiccation they produce; chemical agents by altering the essential composition of the bioplasm, and so interfering with the molecular atomic rearrangements which underlie its exhibitions of energy; and light, heat, and electricity presumably act in a similar way. From the investigation of this subject one or two facts are ascertainable that specially bear upon the subject I am now particularly considering. One is that bioplasm,

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far from being a material uniform in composition and characters, exhibits very great variety, not only in the cells of different tissues, but in the same cell at different times and under different circumstances. Evidence of this is seen in the various behaviour of cells to staining reagents; and, indeed, different parts of the same cell respond to these different dyes in a different way. Still more striking, as showing the variable composition and constitution of bioplasm, are the effects of poisons, some of which are quite innocuous to some cells whilst violently toxic to others, and the greatest variability exists in the degree of resistance offered by different cells, whether they be unicellular organisms or the component cells of the tissues in the highest forms of life. Indeed, the therapeutic value of many drugs—certainly of those whose use is based upon their known physiological action—depends upon the selective capacity of certain tissues as contrasted with others. A still more important point bearing upon this subject, which has come out in the course of experiments on bioplasm, is that within certain limits the resistance of the living matter may be altered in such a way that agencies which ordinarily would be prejudicial to its activities, or even fatal, might be submitted to with impunity. This fact of adaptability to its surroundings, this ability to become acclimatised, so to say, we speak of as immunity when the agent is a toxic one. But the same results are to be found when the disturbing factor is heat, light, or electricity.

Another interesting phenomenon in connection with the effect of external conditions acting within certain limits upon bioplasm, is the determining of the direction of movement exhibited by living matter, upon which they exert an attractive or repellent effect—one form of this we are familiar with as chemiotaxis in states of leucocytosis, but it is probable that other forms of "taxis," caused by heat, light, &c., also prevail—for experiments with unicellular organisms and even some more complicated clearly suggest such to be the case.

Ultimately, no doubt, the mode of action of these agencies, whether this be in a direction favourable to the maintenance of the organism and to its satisfactory development, or as tending to affect it to its harm, is a question of interference with metabolism, a term which is expressive of the constant interactions that take place between the protoplasm and its environment, and of the interactions among the constituents of the protoplasm. But

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inasmuch as one of the parties to this interaction is the living organism itself, which is of very variable nature and capable of artificial alterations in its behaviour, it is evident that the living matter is partly responsible for the result that any external agent, good or bad, produces. And, further, to be satisfied with merely ascertaining the external condition which has led to any condition of disease, is to take a very narrow view of ætiology; whilst to endeavour to throw the whole responsibility for the ultimate results upon this external condition is neither scientifically accurate nor practically sound. It can scarcely be urged in any such assembly as this, "What have we to do with bioplasm and cells, we want to recognise the signs and symptoms of the maladies we are called upon to deal with, and, if possible, to cure them—or at least to treat them." Inasmuch as the first object of our art is to prevent disease, and the final pursuit of pathological inquiry is to ascertain the precedent causal conditions, anything that assists in forming a more accurate conception of the nature of the various causes and their modes of action should be welcomed, even if the practical value be not apparent on the face of it. And whilst we at once accept poisons, extremes of heat and cold, and the like, as the causes of disease, it is in their action upon the living constituents of the tissues that they really produce their effects, and the more precise we can be in locating their results the more likely we are to be successful in applying their antidotes or in averting their consequences.

In attempting to comprehend, therefore, the effects of external agencies, however simple or however complicated they may be, we have also to reckon with the responsive capacity of the living organism. Thus it is that we see the various effects of the same untoward circumstances upon different individuals. How it is, for instance, that the exposure to cold and wet will, in several persons similarly circumstanced, bring about very different forms of disease; whilst the one develops an acute nephritis, another is attacked with acute pneumonia, another falls ill with what is known as a rheumatic attack, and a fourth maybe suffers not at all. More precisely the differences exhibited by individuals in respect to natural immunity to infection, and still more strikingly the immunity that may be artificially acquired, and perhaps most impressive of all the various forms of response to particular foods, drugs, or other ingesta, known as idiosyncrasy, conclusively demon-

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strate that in modifications of the working of the organism it is not only the disturbing external factor that has to be considered, but also in some measure the organism itself. Great and important as was the discovery of the microbic cause of tuberculosis, I venture to think it will be long before those who have had any extensive experience of the ravages of that disease, will admit that the *Bacillus tuberculosis* is the only thing to be kept in view, either in the causation, the prevention, or the treatment of the malady, and I further presume to affirm that any teaching that fails to take account of the personal or intrinsic factor in the pathology of the disease is contrary to the light and likely to be harmful in its results.

The Organism.

Whilst it is quite possible to investigate the phenomena and conditions which occur external to living things quite apart therefrom—indeed, vast branches of inquiry, such as physics, chemistry, astronomy, are entirely so occupied—it is impossible to deal with living things apart from their environment, nor is the attempt to imagine them so isolated in any way profitable. It is as the resultant of the interactions between a complex substance and the conditions around it that we find life, and with the cessation or grave disturbance of these interactions life ceases and death occurs. The consideration of the living organism therefore, apart from these conditions, is inconceivable.

Giving a certain state of the living matter — a state quite indefinable—and a certain state of the environment, the resulting interactions are recognised as healthy, and these natural states are regarded as satisfactory. Variations in these states are possible, within limits, without any recognisable ill effects, but with departures from a certain standard of environment perverted interaction—disease—appears. This responsibility for disease on the part of the environment has just been considered, and the limitation of that responsibility pointed out. It now remains to consider whether the organism itself can be said to have a similar responsibility; whether disease is ever wholly intrinsic in origin, or whether it is always dependent in some measure upon externals. In considering this aspect of the question, there would seem to be two directions in which an essentially intrinsic disease might originate—viz., as the result of inheritance, or as self-developed

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after birth; but in each case it resolves itself into an inquiry concerning the character of the material—bioplasm—through which life, whether healthy or morbid, is manifested, and its ability to transmit acquired modifications such as disease is.

There is one characteristic of living matter which, though quite obvious, is apt to be lost sight of, and although manifested under the influence of external conditions, cannot be regarded as essentially dependent upon them. We are in the habit of referring to the living elements of the tissues without due regard to their entirely transient nature. The peculiarly vital manifestations—the various forms of irritability in response to stimulation, the capacity for assimilation of extraneous material, the adaptability within certain limits to external conditions, and the power of reproduction—by which we recognise the presence of bioplasm, are continued until they naturally terminate in death. But the material itself through which these functions are manifested is ever changing, as the exhibition of these various forms of energy is the expression of a perpetual molecular disintegration and renewal. Thus the functional manifestations are continuous, though the underlying structural basis is constantly shifting; the muscle-fibre or nerve-cell of to-day is freshly constituted by to-morrow, though its capabilities remain constant. It is true that the *form* of the substance—the cell, the fibre, &c., persists, but not the actual constituents. We are bound to regard so remarkable a series of phenomena as a continued repetition of similar form with identity of potentiality, but with a perpetual change in material constitution as an inherent attribute of the living matter, for which the environment cannot be conceived to be primarily the cause, however much it may be subsequently involved; and surely the full appreciation of such a complex must suggest the directions in which the organism itself may become responsible for perverted functions, which we recognise as disease. The substantial identity of life under the greatest diversity of environment gives further support to the same contention.

As with other questions of a kindred nature, opinion has undergone many changes at different times. The responsibility of ancestry for the occurrence of disease was, even recently, most extensively invoked. In its extreme application it constituted a form of fatalism and relieved the individual and his surroundings of all, or almost all, share in the causation of disease. It formed

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a convenient way of shifting on to our predecessors the responsibility for conditions the existence of which later knowledge has shown should have been more properly attributed to the environment. At the present day, with the vast impetus that has been given to the investigation of external conditions by the discovery of the bacterial origin of many diseases, there is, to my mind, a fear that the pendulum may swing too far in that direction—and indications of this, I think, are not wanting—with the result that too little attention is paid to the individual, and the share he takes in the origination of the disease, or, what is more important, the part he should be made to play, whether in the prevention of disease or in the treatment of it when existent.

That certain characters, structural and functional, are heritable is a matter of certainty. The offspring closely resembles the parent, though minor differences exist which exclude absolute identity; other differences occur among the offspring themselves, and equally exclude identity in their case. Moreover, this resemblance of progeny to parent is anticipated and relied upon. No one expects a chestnut to grow from an acorn, nor a cat to be developed from the fertilised ovum of a bitch; anything but an oak or a dog respectively would be against universal experience. Nor is it for a moment suggested that this specific similarity of offspring and ancestor is determined by conditions of the environment, or that it is otherwise to be explained than the expression of the potentiality of the blended sexual elements; and the same would be admitted regarding the possession of characters, however trivial or unimportant, that would be clearly accepted as transmitted. But it is a highly disputed question how far, if at all, such somatic modifications as the individual may have acquired can be transmitted to his posterity. It might have been supposed that the innumerable perversions of structure and function manifested in the many forms of disease with which we are acquainted, would have afforded the opportunity of determining this question; but it cannot be said that such is the case, and the subject of the hereditary transmission of acquired characters still divides biologists. The hypothesis of germinal continuity, with which the name of Weissmann is especially associated, offers at least an explanation of a large number of facts, and, what is of great value, affords a comprehensive and intelligible basis upon which thinkers can formulate their points of agreement and difference, and thus permit

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a common ground of argument, where previously all was vague, and much disputation was at cross purposes for want of an accepted definition of the subject. Briefly stated, the hypothesis—and, be it observed, it is little more than an hypothesis, at least so far as being universally applicable—is this:—The germ-plasm of the fertilised ovum, whilst in great part undergoing structural development and differentiation into the likeness of the parent form, remains in small part unaltered, and in some species, even from an early stage, becomes segregated from the rest in the future sexual organs of the adult, whence, in conjunction with similar material from the opposite sex, it goes to form the succeeding generation, in which it develops and differentiates again, leaving a part to be continued on as before. Thus, as has been aptly said, "The parent is rather the trustee of the germ-plasm than the producer of the child, and with similar conditions to start with, and similar conditions in which to develop, therefore like tends to beget like."*

Injury or disease inflicted upon the soma—that is, the part of the germ-plasm which by differentiation and development forms the body of the offspring—will not be transmitted, and as a large number of maladies produce no deeper effect than this, however serious they may be, it is claimed that few diseases are heritable. I need scarcely stay to point out that such affections as appear at or soon after birth, and are the result of infection or damage to the organism whilst developing, are in no proper sense of the word inherited, though they may be conveniently grouped as congenital. Should, however, the disease or injury have affected that portion of the germ-plasm set apart in the individual for the continuance of the species—resting, as it were, in the one generation to develop in the next—then, by the nature of the case, the morbid condition will be transmitted, and hereditary disease occur; and, moreover, it is assumed that, should the malady of the individual be of such a nature as profoundly to affect the general nutrition of the organism to such a degree as to involve the germ-plasm, then the state itself, or maybe some allied one, is propagated. I do not propose to do more than refer to the provisional hypothesis, put forward by Darwin, of pangenesis, and to point out that such a view is the exact opposite to that suggested by Weissmann, inasmuch as the ova and spermatozoa are regarded as constituted of gemmules derived from all the cells and all the tissues of the organism, including any

* Article, "Heredity," by Prof. Arthur Thomson, 'Encyclopædia Medica.'

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part which has undergone any acquired modification, which thereby becomes transmitted along with the other and more specific structural characters of the individual to the resulting offspring; and it suffices to say that, whichever theory be adopted, the responsibility of the organ itself for the transmission of the morbid potentiality is obvious; nor am I concerned to criticise Weissmann's theory, since it is only in its bearing on the part played by the individual in the transmission of normal or abnormal characters that I am at present interested.

If now we turn to consider what bodily states are to be regarded as heritable, we shall find that the number is very considerably less than was formerly supposed, and that the transmissibility, even in those cases which are accepted, is not so potent as was thought. In other words, that it is less the disease itself than a predisposition or tendency towards it, often requiring for the development of the malady a suitable conjunction of external conditions, in the absence of which the individual escapes; though this cannot always be so, for the tendency may be so strong as to assert itself in spite of the most favourable environment.

Prominent among those conditions which tend to recur in successive generations are longevity and the reverse. Statistics have shown that the life duration is a particularly family characteristic, and reduced to its physiological terms this represents a fundamental quality of the living material of the body—bioplasm—together with the power of resistance or susceptibility to the effect of untoward surroundings.

States which insensibly depart from the healthy type, reaching to a degree in which the well-being is interfered with, are represented by an undue formation of fat, obesity, or an exceptional leanness, and such states are notoriously prone to characterise the members of families over many generations.

A third group of morbid states which recur with sufficient frequency among relatives of successive generations comprise such essentially nutritive disturbances as diabetes and gout, and it is peculiarly noticeable that maladies of this class do not invariably "breed true," but one or other term of the series, in which is to be included obesity, may represent the underlying malnutrition.

Perhaps even more marked illustrations of disease appearing in the offspring, similar to those from which the parents suffer, are to be found among nervous maladies. The frequent transmission of

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such conditions is notorious. But a careful examination shows that, whilst it is comparatively seldom that any special nervous affection, such as chorea, tabes, or apoplexy, occurs in members of the same family, what is far more commonly transmitted is some form of general nervous disturbance or undue susceptibility, such as neurasthenia, acting upon which unhealthy surroundings—*e.g.*, syphilis, alcoholism, mental fatigue, or anxiety—may determine some specific nervous disease.

Lastly, there are some affections which are but imperfectly understood, such as hæmophilia and colour-blindness, which exhibit a remarkable liability to transmission, and curiously the former is chiefly manifested by males, whilst transmitted by females.

It is apart from my present purpose to attempt any exhaustive account of diseases that we consider as hereditarily propagated, from the frequency with which we observe their recurrence in successive generations, and it is probable that the list may even be shorter than it now is, as extended knowledge may show the absolutely essential requirement of some external factor for their manifestation. But so far as our present ideas go, I think I should find general agreement in assigning those I have mentioned to the class of hereditary diseases, leaving it an open question how far the predisposition is of itself sufficiently potent to determine the occurrence of the disease, or how far any environmental circumstance is requisite to develop it.

As further illustrating the responsibility of the organism itself in the occurrence and manifestation of disease may be mentioned the undoubted influence exerted by age and by sex. Even when all allowance is made for the varied external conditions attendant upon these states, there still remains a sufficient difference in the incidence of disease at different ages—that is, the difference in susceptibility—and also in the manner in which morbid states manifest themselves. The far greater frequency of the acute infections in the young as compared with adult or old age, the diseases which characterise the periods of growth and those of decline, will serve to exemplify the one, as the far greater frequency in females of neuritis as a sequence of alcoholism, or of a mitral obstructive lesion in the same sex, than in males, will illustrate the other.

A still more conclusive example of the responsibility of the organism is to be seen in the recovery from disease. A large

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number of the morbid states tend of themselves to revert to the normal; the disturbed metabolism swings, slowly or quickly, back to its healthy course, unless the damage it may have inflicted is too serious or profound. It is true that the completeness of recovery, or even recovery at all, may be hindered and retarded by unfavourable external conditions, but the potentiality of amendment is essentially intrinsic, and of this we take advantage when we adapt our therapeutic measures in such a way as to put our patients in the most satisfactory position for self-cure.

Upon the intrinsic nature of the living matter itself, so far at least as the germ-plasm is affected (whether this be so inherently or subsequently by such changes in the soma as shall have reacted upon or involved it), must be dependent certain departures from the normal state, just as that normal state itself is primarily dependent upon the intimate molecular constitution of the bioplasm. But it must be admitted that it is more frequently a predisposition or tendency that is transmitted which may require for its development a favourable conjunction of the environment. The essential responsibility of the organism, however, remains undoubted. Equally so does this responsibility underlie the principle of variation which determines the characters of the individual as opposed to the characters of the species, though this again may depend in some measure upon externals for its exercise and for its perpetuation. Nor can the environment be credited with the production of those atavistic characters that represent latent or dormant ancestral qualities, nor the transmission to the offspring of the second male parent characteristics derived from the first, the phenomenon that is known as telegony, and upon the reality of which much doubt has recently been cast.* It must be the organism itself that is fundamentally responsible for their appearance, however some external factor may appear to have evoked them, and whilst these traits may without difficulty be conceived of as becoming manifest solely by intrinsic conditions of the organism, the environment alone can by no stretch of imagination be regarded as alone provocative of them.

Now, it appears to me that just as we recognise that predisposition to disease rather than the disease itself is transmitted, a vulnerability of tissue that permits of an easier disturbance by

* *See* Prof. Cossar Ewart's Address in the Section of Zoology, British Association for Advancement of Science, 1901.

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external circumstances than normal, and that such predisposition or tendency is a property of the organism, so do we require some readjustment of our terminology, and, though I am aware of the reluctance of some minds to pursue a precision in expression, I feel emboldened to advocate the use of certain terms, and the more so as they are not novel in themselves, nor originated by me, though I would strongly urge a consistency in their application.

In the word *constitution* we have ready to hand a word that may be taken to signify the sum total of the tendencies possessed by a living organism whencesoever derived, and whether beneficial or otherwise. But inasmuch as we must recognise that as there are variable forms of unhealthy constitutions which may be fitly termed *diatheses*, and even variable ways of being ill with the same malady, so there are several forms—I hesitate to say types—of healthy constitution, several ways, that is, of being well, for which the old word *temperament* seems properly applicable. And this, however much we may differ among ourselves as to the structural characteristics of these temperaments, or the influence they may exert in predisposing to disease. Essentially connected with these expressions is the idea of persistency, and they should not be applied to any condition, whether of health or disease, which is expected to pass away and leave no trace.* With changing views as to the nature of disease and our extended knowledge, these words have undergone considerable alteration in the meaning attached to them, or they have been discarded altogether. Thus it is that "temperament" has come largely to denote certain mental characteristics which have some affinity with the physical conditions now referred to. Some there are who have refused to see any connection between different types of health and special proclivity to disease, and are not a little sceptical as to the reality of such types.† But it seems to me difficult to doubt their existence if hereditary predispositions are granted, though how far susceptibilities to disease are associated with the structural characteristics of normal temperaments precise observation alone would determine.

And here I would digress a moment to say how much I wish this Society could see its way to prosecute some collective inquiry of this

* 'The Pedigree of Disease,' by Jonathan Hutchinson, F.R.C.S., F.R.S., 1884.

† "The Physiognomy of Disease," by Prof. Gairdner, in Finlayson's 'Manual of Clinical Medicine.'

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kind, the ascertainment of clinical observations contributing to the settlement of undetermined questions. Other societies have done so with success, and the wide field of experience covered by members of the Medical Society of London should be sufficient if properly directed to furnish some valuable information, and I know of no subject that would be more interesting and worthy of such investigation than the incidence of disease in successive generations.

I have now shown, obviously with no pretence to originality, that whether the occurrence of disease be primarily determined *ab extra*, or its starting point be essentially intrinsic, however much favoured by externals, the organism itself is in part responsible, and that that responsibility cannot be disregarded. In what I have said I have tacitly assumed the physico-chemical explanation of life which has been generally accepted for the past half-century, replacing as it did a vitalistic theory that in one or other of its many aspects had previously occupied the field for so long. At the present day, however, any one dealing, as I have imperfectly done, with the fundamental questions which lie at the root of all biological knowledge is bound, if only for completeness, to make some reference to that recrudescence of vitalism in yet another aspect, which a small but distinguished body of biologists have recently fostered. Dissatisfied with the physico-chemical hypothesis as inadequate to explain vitality, though recognising the enormous benefits it has conferred in the prosecution of physiological inquiry, they have endeavoured to show not that the older vitalistic conceptions should be reinstated, since they also are far from satisfying, though not without suggestion, but rather what has been termed a "neo-vitalism." This seeks to show that "the biological are separated from the physical sciences, not through the existence of any spectral line of demarcation between what is living and what is not living, but by the fact that the fundamental conceptions of biology are, and from the nature of the phenomena dealt with, must be, entirely different from those of physics and chemistry." The direction in which the explanation of these phenomena is to be found is in the pursuance of a method similar to that "which the morphologists have so successfully applied to the elucidation of structure." The ground idea of the new anatomy was that of the existence of an immanent type, a plan which an organism or group of allied organisms adheres

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to through every variety of outward modifications. Adherence to morphological plan is evidently not the same thing as persistence of physical structure or configuration; for the physical configuration may vary enormously in cases where identity of morphological plan is perfectly clear. "The fundamental assumption of morphology is that each part of an organism is determined as regards its mode of existence by its relations to the other parts. . . . The results of embryological experiments illustrate very clearly the fact that the parts of an organism mutually determine one another's mode of existence. Now, this implies a constant physiological determination of the nutrition of each part in accordance with the morphological plan of the whole. The morphological conception of an organism is thus just as much a physiological as an anatomical conception. As, however, the nutrition of an organism depends not merely on the relation of the parts to one another, but also on their relation to the physiological environment, it seems equally clear that the conception in question implies that the influence of the environment also is determined in a similar manner to that of the parts of the organism. We are accustomed to look at environment simply in its physical and chemical aspects. It must be remembered, however, that what the environment is for an organism depends on the organism itself, and that the influence on the organism of any particular physical or chemical condition in the environment may vary indefinitely according to the nature or physiological state of the organism."*

It is not my intention, even were I capable of so doing, either to support or to oppose these conceptions, and my purpose in making these quotations from the writings of one who is foremost in their advocacy is fully served by showing that if the effect of the environment on an organism is conditioned by the physiological state of that organism, then the responsibility for disease must be much greater on the part of the organism than I have ascribed to it. And I would add that some of the phenomena of disease appear to me strongly to support that view. It would certainly accord with the difference in result that follows the infliction of some detrimental external agency upon several healthy individuals similarly situated, and is consistent with the facts of immunity or of increased

* "Vitalism," by Dr. Haldane, F.R.S., 'Nineteenth Century,' September, 1893.

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susceptibility, and also with the differences manifested during the course of the same disease attacking different persons.

I am aware how foreign to the general work of this Society is the subject that I have brought to your notice, and yet I feel ill-disposed to apologise for having taken advantage of my position on this occasion to treat of so theoretical a matter, however much I might do so for the triteness of my theme. For I hold that for the intelligent pursuance of our art, and for the clearer conception of the many problems it offers to us, we require some guiding principles, be they only hypotheses to be thrown aside as greater knowledge renders them untenable. I know how sternly matter-of-fact is the character of this Society, and how the results of observation or of practice meet here with a criticism that goes far to establish their results as facts of our science, and how strongly mere speculation is discouraged; yet, knowing this, I have dared to quote from a comment on the recent address of the distinguished President of the British Association for the Advancement of Science, who had made the atomic theory and its claim to continued support the subject-matter of his remarks, I would say:—"Facts, if they be well ascertained, are always worth seeking and possessing, yet little real progress can be made while they are merely accumulated and are considered separately or in small groups. Knowledge can only accrue when they are regarded in the relations in which they stand to one another, and as perfectly accountable results of a fundamental law. This is so true that a wrong theory, as long as it enables phenomena to be considered in connection with one another, may be at times a more useful instrument than one which is accurate but more limited in scope."

Without giving full assent to the last statement, I am convinced of the wisdom of the general proposition, and I am a little inclined sometimes to think that in our steady avoidance of theory we sometimes, to our own detriment, miss a useful guide, and fail in taking a comprehensive view of our subject, so long as we hesitate to recognise the bearings and relations of the facts we so highly prize and strenuously seek after.

"*Observation*, however accurately performed, does not add to our *comprehension* of Nature, unless it is controlled by a scheme of thought for which confirmation or disproof is sought in the results of the observation. Some principle already shaped, whether it be a product of our own mental activity, or be handed on to us by

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authority, is wanted to give to each fresh item of perception a rank in an orderly plan, and to add clearness and value to that which would be, otherwise, unrelated and imperfect."*

But in endeavouring to form some estimate of the share taken by the individual in the determination and manifestation of disease, we are dealing with a subject that is essentially practical in its bearings, however speculative may be the basis which subtends the application. The correct estimation of the value of the facts ascertained when inquiring into the family history and life history of our patients, depends upon some knowledge of the workings of the organism in response to externals, and of such inherent qualities and capabilities he may possess. Whilst it may seem but little towards the framing of a diagnosis in the use of that term which is restricted to the mere naming of the disease, in a wider sense of comprehending the real nature of the case it is all-important, and underlying, as it does, prognosis, the estimate of the individual resistance to the disease, and furnishing thereon the only rational plan of treatment, its study cannot be too sedulously cultivated. I would claim, therefore, that I have not, after all, wandered far afield from our legitimate occupation, and whilst *dulce est desipere in loco* I venture to hope my dissipation in generalities has not been out of place.

* 'The Living Organism,' by A. Earl, M.A., 1898.

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October 28th, 1901.

TYPHOID FEVER IN SOUTH AFRICA.

By A. ELLIOT, M.A., M.D. Edin., M.R.C.P. Lond., D.P.H. Lond.; and J. W. WASHBOURN, C.M.G., M.D., F.R.C.P. Lond., F.R.C.S. Eng.

A LARGE number of the cases of typhoid fever admitted into the Imperial Yeomanry Hospital at Deelfontein during the year from March, 1900, to March, 1901, were convalescent on arrival, and the notes of others have been lost. We have, however, collected 262 cases, all of which were in the acute stage when in the hospital, and of which detailed notes were available. We have analysed these cases and have compared them with the following:—1. A series of cases which were admitted into the emergency hospital during the epidemic of enteric fever at Maidstone in the year 1897. These cases, 210 in all, have been carefully analysed by Dr. T. B. Poole.* 2. Statistics of 611 cases analysed by Dr. F. F. Caiger and Dr. E. W. Goodall.† 3. An analysis by Dr. W. Osler ‡ of 829 cases treated in the Johns Hopkins Hospital during the years 1889 to 1899. 4. Statistics of 506 cases at the Eastern Fever Hospital during the years 1892–1894.§ 5. Other accounts of typhoid fever during the campaign by Dr. H. H. Tooth, Dr. H. D. Rolleston, and Dr. F. D. Boyd. Our chief object has been to point out the main features of the typhoid fever occurring in South Africa and to ascertain whether it differs from that met with in England and America. At the same time we have considered the influence of inoculation upon the course and the incidence of the disease, and we have given a brief account of a few cases of special interest.

Mortality.—There were 36 deaths in our 262 cases, or a mortality of 13·7 per cent. At the Metropolitan Asylums Board Hospitals in 15,632 cases during the years from 1871 to 1900 the mortality was 18·44 per cent., while in 1900 the mortality was 14·09 per cent. In the Worthing epidemic in 1894 there were 2,257 cases, with a mortality of 13 per cent. During the Maidstone epidemic 1,847

* 'Guy's Hospital Reports,' vol. liii.

† 'Metropolitan Asylums Board Reports,' 1896.

‡ 'Johns Hopkins Hospital Reports,' vol. viii, Nos. 5 to 9, 1900.

§ Quoted in Goodall and Washbourn's 'Manual of Infectious Diseases.'

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cases were notified, with a mortality of 7·14 per cent. Dr. Osler in his text-book gives a mortality of 11·2 per cent. at the Montreal Hospital for 20 years, and at the Johns Hopkins Hospital a mortality of 7·5 per cent. in the 829 cases analysed. At the Edinburgh and East of Scotland Hospital in South Africa the mortality was 10·7 per cent. In considering mortality the age and condition of the patient must be borne in mind. Most of our patients were young adults, in whom the disease is less severe than in older people, but more severe than in children, and many of them had suffered from considerable fatigue and privation not only before but even after they had been attacked. Our mortality (13·7 per cent.) is practically the same as that of the Metropolitan Asylums Board Hospitals during 1900, and as that of the Worthing epidemic; it is almost double that of the Maidstone epidemic and the Johns Hopkins Hospital cases.

Relapses.—Sixty-one of our 262 patients suffered from relapse, but in 36 of these relapse arose either immediately or shortly after admission, the primary attack having occurred elsewhere. Subtracting these cases, there remain 25 out of 226 patients who relapsed in the hospital, an incidence of 11 per cent. Dr. Osler, in 829 cases at the Johns Hopkins Hospital, gives an incidence of 10 per cent.; Dr. Goodall and Dr. J. W. Washbourn, in 506 cases at the Eastern Fever Hospital, an incidence of 13 per cent.; Dr. Caiger and Dr. Goodall, in 611 cases, an incidence of 11·9 per cent. Dr. Rolleston, in an analysis of 244 cases at the Imperial Yeomanry Hospital at Pretoria, gives an incidence of 21 per cent. He, however, informs us that there are included among his cases a certain number of patients who had suffered from the primary attack elsewhere, which, we think, will account for the high incidence. At the Edinburgh and East of Scotland Hospital in South Africa in 150 cases the incidence was only 5·3 per cent. Possibly this low rate may be due to the early transfer of cases to other hospitals. It will thus be seen that our incidence of relapse corresponds with the average in England and America. We have no doubt that the too early removal of patients predisposes to relapse. Thirty-six of our cases relapsed either immediately or shortly after admission, which we believe was due in many cases to too early removal and improper food on the journey. Of the 25 cases of relapse the shortest interval that elapsed between the end of the primary attack and the commencement of the relapse was one day; the longest interval was

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31 days; the average being 10·8 days. In Dr. Poole's cases the longest interval was 24 days, and the average nine days. Of 53 cases of Murchison quoted by Dr. Osler the average interval was 11 days. In the Johns Hopkins Hospital cases the longest interval was 36 days, the average being 8·2 days. The shortest duration of the relapse was six days, and the longest 23 days, the average duration being 13·5 days. In Murchison's cases the average was 15 days. Two of our cases suffered from two relapses, and one from three. We had no deaths among the 25 cases of relapse included in these statistics, which bears out the general experience of low mortality among relapses.

Second attacks.—In 12 of our 262 cases there was a history of a previous attack, an incidence of 4·5 per cent. There were actually only 99 cases in which it was definitely stated in the notes whether the patient had or had not suffered from a previous attack. This would give a much higher incidence of second attacks, but as the fact of a previous attack not having occurred might not have been noted we prefer to accept the percentage upon the whole of the cases. Dr. J. Dreschfeld in his article in Clifford Allbutt's 'System of Medicine' states that at the Hamburg General Hospital, out of 2,000 cases there were 14 in which there had been one previous attack, and one case in which there had been two previous attacks, or an incidence of 0·75 per cent.; while Berg only found one second attack in 1,624 cases, or an incidence of 0·06 per cent. Our incidence is, therefore, extremely high, and this appears to correspond with the experience of others who have worked in South Africa. Brigade-Surgeon-Lieutenant-Colonel Alexander Crombie,* for instance, in the discussion upon Dr. Tooth's paper at the Clinical Society of London, states that of 150 officers who had returned invalided from South Africa for enteric fever 6 per cent. had suffered from previous attacks. The frequency of second attacks in South Africa appears to be chiefly due to the intensity and frequency of exposure to infection. In England individuals are probably not often exposed to infection. The fatigue and privation experienced in South Africa would also be a predisposing factor in rendering individuals liable to infection. One of our patients had suffered from two previous attacks and died during the third. The remaining patients recovered. The following is a short account of these cases:—

* 'The Lancet,' March 30th, 1901, p. 933.

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No. 28.—The previous attack occurred seven years ago. The present attack was followed by relapse. The patient recovered.

No. 84.—The patient was an officer. The date of previous attack was not stated. The patient was admitted towards the termination of the attack, which was followed by empyema. The patient recovered.

No. 103.—The previous attack was 15 years ago. The present attack was attended by diarrhœa, spots, and enlargement of the spleen. The patient recovered.

No. 104.—The previous attack occurred when the patient was a child. The present attack was attended by enlargement of the spleen and by spots, and was complicated by otitis media. The patient recovered.

No. 160.—The patient was an officer. The previous attack occurred three or four years ago. The present attack lasted for 26 days, and gave the serum reaction. The patient recovered.

No. 206.—The patient had had one previous attack as a schoolboy, and another during the campaign. The present attack was very severe, and proved fatal.

No. 222.—The previous attack occurred one year ago. The present attack was attended by enlargement of the spleen and spots. The patient recovered.

No. 232.—The previous attack occurred in India 11 years ago. The present attack was mild. The patient recovered.

No. 243.—The previous attack occurred three years ago, and the patient had been inoculated three times just before the onset. The previous attack was due to the accidental ingestion of the typhoid bacillus in experimental work. The present attack was severe. The patient recovered.

No. 279.—The previous attack occurred in Egypt eight years ago. The present attack was accompanied by spots. The patient recovered.

No. 300.—The previous attack was in April, 1900. The patient was inoculated in January, 1900. The present attack was sharp, and was accompanied by enlargement of the spleen, by diarrhœa, and by copious spots. The patient recovered.

No. 54.—The previous attack occurred four or five years ago. The present attack was followed by definite relapse. The patient recovered.

The influence of inoculation upon the course of the disease.—In 211 of our cases it was definitely recorded whether the patient had been inoculated or not; 186 of these cases had not been inoculated, with 20 deaths, or a mortality of 10·7 per cent.; 25 had been inoculated, with four deaths, or a mortality of 16 per cent. The mortality was thus higher among the inoculated than among the non-inoculated. At the Portland Hospital 54 cases were inoculated, with four deaths, and 178 not inoculated, with 25 deaths. At the Imperial Yeomanry Hospital in Pretoria there were 27 cases inoculated, with no deaths; and 115 cases not inoculated, with six deaths. At the Scottish Hospital there were 15 inoculated, with one death; and "about" 77 not inoculated, with 10 deaths. At the Edinburgh and East of Scotland Hospital there were 18 cases inoculated, with no deaths, but it is not definitely stated how many

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patients died among the non-inoculated. Adding together the cases at Deelfontein, Pretoria, the Scottish Hospital, and the Portland Hospital we find that there were in all 121 cases inoculated, with nine deaths, a mortality of 7·4 per cent.; and 556 not inoculated, with 61 deaths, a mortality of 10·9 per cent. In dealing with such small numbers the difference between 10·9 per cent. and 7·4 per cent. is too slight to point to any marked beneficial influence of inoculation upon the course of the disease. In our own series the mortality is greater among the inoculated than among the non-inoculated.

The influence of inoculation upon the incidence of the disease.—In our staff there were 44 nursing sisters: of these, 40 were inoculated and two contracted enteric fever, four were not inoculated and none contracted the disease. There were 10 ward-maids, all of whom were inoculated and one contracted enteric fever. There were 20 medical officers: of these, nine were inoculated and one contracted enteric fever; 11 were not inoculated, and none of these contracted the disease. There were 10 dressers, none of whom were inoculated and four contracted enteric fever. Taking the staff altogether, there were 59 inoculated, of whom four contracted enteric fever, and there were 25 not inoculated, of whom four also contracted the disease. It will be noted that the four of the non-inoculated who contracted the disease came from among the dressers who were younger than the rest of the staff. At the Irish Hospital, quoted by Professor A. E. Wright, 64 of the staff were inoculated and two contracted enteric fever; four were not inoculated and none of these contracted it. At the Imperial Yeomanry Hospital, Pretoria, 35 persons were inoculated and six contracted enteric fever; 115 were not inoculated and 16 contracted the disease. At the Portland Hospital 28 were inoculated and seven contracted enteric fever; 13 were not inoculated and three contracted the disease. At the Edinburgh and East of Scotland Hospital all—namely, 58—were inoculated and nine contracted enteric fever. Taking all of these together, we find that there were 244 inoculated, with 28 cases, an incidence of 11·4 per cent., while there were 157 not inoculated, with 23 cases, an incidence of 14·6 per cent. The difference between the incidence of enteric fever amongst the inoculated and the non-inoculated is too small to be ascribed to any protective influence exerted by inoculation. We have not included the statistics of the Scottish Hospital, as the exact number of inoculated and

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non-inoculated is not definitely stated. The not infrequent occurrence of second attacks of enteric fever renders us doubtful of the prospect of successful inoculation in the future. The considerable risks of contracting enteric fever incurred in hospital work during this campaign are well exemplified by the above figures. Of a total of 401 persons engaged in attending the sick, 51 suffered from the disease, an incidence of 12·7 per cent.

Hæmorrhage.—We have included under hæmorrhage only those cases in which several ounces of blood were passed. There were in all 16 cases, an incidence of 6·1 per cent. At the Imperial Yeomanry Hospital in Pretoria there was an incidence of 8·7 per cent.; in Dr. Osler's cases 6 per cent.; in Dr. Goodall and Dr. Washbourn's cases 7·9 per cent.; in Dr. Caiger and Dr. Goodall's cases 8·6 per cent.; and in the Maidstone cases 5·5 per cent. Our incidence appears thus to be about the average. Of our 16 cases eight died, a mortality of 50 per cent., and five others were noted as being exceedingly severe cases. Dr. Rolleston states that of 21 cases at the Pretoria Hospital 16 died, a mortality of 76 per cent. It thus follows that in South Africa hæmorrhage in enteric fever is a symptom of grave signification. In England and America the mortality does not appear to be so high; in Dr. Poole's cases there were two deaths, a mortality of 20 per cent., and in Dr. Osler's cases the mortality was 10 per cent. In the majority of our cases —namely, in 13 out of 16—the patients suffered from diarrhœa.

Phlebitis.—There were 15 cases of phlebitis, an incidence of 5·6 per cent. At the Edinburgh and East of Scotland Hospital the incidence was 6 per cent. Lieutenant-Colonel Crombie states that 25 per cent. of his cases suffered from phlebitis, but this high incidence appears to be due to the fact that many of his cases were invalided home on account of the supervention of this complication —a circumstance which vitiates his statistics. In Dr. Poole's cases the incidence was 3·8 per cent., in Dr. Caiger and Dr. Goodall's cases 3·4 per cent., and in Dr. Osler's cases 1·9 per cent. It thus follows that the incidence of phlebitis in South Africa is greater than in England or America. In our cases the left leg was affected 10 times, the right leg four times, and both legs twice. The phlebitis ensued during convalescence eight times, during the pyrexial stage of the primary attack four times, during a relapse twice, and in one case the date was not noted. When occurring during the pyrexial stage it was towards the end of that period, the

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earliest appearance being on the sixteenth day. The latest date of its appearance after the end of the pyrexial stage was 30 days. It is probable that some cases developed phlebitis after leaving the hospital, but of these we have no record.

Pneumonia.—There were 10 cases of pneumonia, an incidence of 3·05 per cent. In Dr. Caiger and Dr. Goodall's statistics the incidence was 3·2 per cent., in Dr. Osler's statistics it was apparently 2·05 per cent., at the Pretoria Hospital it was 2·2 per cent., and at the Edinburgh and East of Scotland Hospital it was 1·3 per cent. The incidence thus appears to be about the same as in other statistics. Of our 10 cases five died, a mortality of 50 per cent., and at the Pretoria Hospital there was a similar mortality—namely, two deaths out of four cases.

Pleurisy.—There were 10 cases of pleurisy, an incidence of 3·05 per cent. In one of these cases there was effusion, which resolved, and in another case an empyema developed which had to be operated upon. In the Maidstone epidemic the incidence of pleurisy was 2·2 per cent. and in Dr. Caiger and Dr. Goodall's cases it was 2·6 per cent.

Bronchitis.—Bronchitis was not a marked feature of the attack. In eight cases it was sufficiently severe to be specially noted.

Periostitis.—There were six cases of periostitis, an incidence of 2·2 per cent. In the Maidstone cases the incidence was 5 per cent.; in Dr. Caiger and Dr. Goodall's cases it was 1·1 per cent. In three of the cases the tibia was affected, in one the femur, in one the index finger, and in one the transverse process of the atlas. They all occurred during convalescence, except one case, which occurred at the end of the pyrexial stage. The case in which the atlas was affected was an interesting one. A painful swelling appeared in the parotid region, which was at first considered to be a parotitis. When opened by Mr. H. A. Ballance the transverse process of the atlas was felt to be bare. The patient made a good recovery.

Condition of the bowels.—Of the 234 cases in which the condition of the bowels is clearly stated, in 100 there was constipation, in 84 diarrhœa, in 41 alternate constipation and diarrhœa, and in nine the bowels were regular. It will thus be seen that in 42·7 per cent. cases there was constipation throughout the attack. Dr. Poole states that 50 per cent. of his patients at Maidstone suffered from constipation, and Dr. Osler, in his analysis of 829 cases at the Johns Hopkins Hospital, states that 34 per cent.

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suffered from constipation. In the Edinburgh and East of Scotland Hospital 37 per cent. suffered from constipation. From these figures it would appear that constipation, not only in South Africa, but also in England and America, is much more common than is usually imagined. In the severe cases diarrhœa was most frequent, and of 35 of our fatal cases there was diarrhœa in 29, constipation in four, and alternating constipation and diarrhœa in two.

Perforation.—There were six cases of perforation—an incidence of 2·2 per cent. Dr. Dreschfeld gives an incidence of from 2·5 per cent. to 3 per cent.; Dr. Caiger and Dr. Goodall, 4·9 per cent.; Dr. Goodall and Dr. Washbourn, 3·1 per cent.; Dr. Osler, 2·7 per cent.; and at the Edinburgh and East of Scotland Hospital the incidence was 2 per cent.

Cardiac complications.—In many cases the pulse was exceedingly rapid, and the patient suffered from cardiac collapse out of proportion to the severity of the attack. We attributed these cases to previous fatigue. Dr. Rolleston calls attention to the rapidity of the heart occurring during convalescence in patients who have been over-fatigued before the attack. We have also noticed the same during convalescence.

Convulsions.—In two cases there were well-marked convulsions, one of which we attributed to the administration of strychnine.

CASE 1.—The patient died at the end of the second week after a severe attack. On the day preceding death there were pharyngeal spasm and retraction of the head. Nasal feeding, which was attempted, brought on general convulsions. No necropsy was made.

CASE 2.—The patient died on the fifteenth day of the attack. On the eighth day there were general convulsions with opisthotonos and trismus, which subsided under morphia and chloroform. He had been taking for the previous 36 hours 5 minims of solution of strychnine every four hours. We attributed the convulsions to the strychnine. (Dr. Osler has described cases of convulsions occurring during enteric fever.)

Abdominal pain.—In 15 cases a special note was made of the occurrence of abdominal pain during the attack. The pain was at some time so acute as to suggest perforation, and, indeed, in one case the abdomen was opened under this belief. The peritoneum was found to be perfectly healthy, and the patient made a good recovery. Most of these cases appear to be simple colic. It is possible that in some of them there was slight peritonitis. Dr. Caiger

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and Dr. Goodall state that in 2·2 per cent. of the cases analysed by them the patients suffered from peritonitis without perforation.

Bladder trouble.—Difficulty in micturition was noted in five cases at a time when the mental condition of the patient was perfectly good, and in some cases quite early in the disease. In one of the cases a catheter had to be used all through the attack, and in three cases on several occasions. In two other cases there was distinct cystitis. This list does not include the cases in which retention occurred while the patient was unconscious.

Parotitis.—There were three cases of parotitis, an incidence of 1·1 per cent. Dr. Caiger and Dr. Goodall gave an incidence of 0·6 per cent., Dr. Poole an incidence of 0·5 per cent., Dr. Osler an incidence of 1·4 per cent., and at the Pretoria Hospital the incidence was 1·1 per cent. In one of our cases the parotitis was double and suppurative; the patient died. The other two cases were unilateral, one being suppurative and the other non-suppurative; both recovered.

Pharyngo-tonsillitis.—There were 10 cases, an incidence of 3·05 per cent. In some of the cases there was exudation upon the tonsils of a follicular character.

Abscesses.—In one case two small abscesses developed, one in the right inguinal region and the other in the peri-anal tissue.

Gangrene of the skin.—In one case local necrosis of about the size of a florin occurred in two places—on the anterior surface of the right thigh and over the right tibia. The patient recovered.

Appendicitis.—In two cases enteric fever immediately followed a definite attack of appendicitis. In one case the patient recovered, but the other was fatal. *Post-mortem*, adhesions around an appendix distended with muco-pus were found in addition to the typhoid lesions. In another case, not included in our statistics, suppurative appendicitis followed an attack of enteric fever. Recovery ensued after operation. In the Edinburgh and East of Scotland Hospital a mild attack of appendicitis supervened in one case.

Eruptions.—In some of the cases the typhoid eruption was exceptionally profuse. In two cases a scarlatiniform eruption occurred which was attributed to an enema. In two cases there was a distinct *tache bleuâtre*. In six cases purpuric spots appeared on the skin, especially on the limbs; three of these cases were associated with spongy gums, and in one case there was frequent and profuse hæmorrhage.

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Secondary fever.—Temporary rises of temperature occurred in a large number of cases. In many of these cases the fever appeared to be due to constipation.

Otitis media.—There were four cases, an incidence of 1·5 per cent.

Pericarditis.—There were three cases, an incidence of 1·1 per cent.

Hepatitis.—In one case there was hepatitis. The patient had a great and uniform enlargement of the liver during the pyrexial period. Abscess was suspected, but the enlargement subsided with the defervescence. The patient had not suffered from dysentery previously. Dr. Rolleston mentions a case of solitary liver abscess following enteric fever in a patient who had not previously suffered from dysentery, and who recovered after operation. Dr. Osler reports a case of enlargement of the liver which subsided and ended in recovery.

Jaundice.—This occurred in only one case, and was associated with fatal pneumonia.

Orchitis.—There were no cases of orchitis.

Dysentery.—There were 21 cases of dysentery occurring either during the attack of enteric fever or shortly before or after it. In six of these cases the dysentery preceded the attack, and all of the patients recovered; in four cases the dysentery occurred during the attack, and two of the patients died; while in 11 cases the dysentery occurred during convalescence, and four of the patients died. It would thus appear that dysentery arising either during the attack or during convalescence is of serious signification, the mortality being 33·3 per cent.

Malaria and rigors.—In 11 cases definite rigors occurred during the attack. In two cases the malaria plasmodium was found, and probably was present in a third, the patients at the same time giving the serum reaction. Dr. Poole noticed rigors in eight of his cases, an incidence of 3·8 per cent., and Dr. Osler has specially called attention to their occurrence. Omitting the definite malaria cases, our incidence was 3·05 per cent. The following is a history of the 11 cases:—

Case 1.—This was a severe and prolonged attack of enteric fever, the pyrexia lasting for nine weeks. During the fourth week there were daily rigors without apparent cause. The patient was attacked with phlebitis three weeks after the first rigor. The patient recovered.

Case 2.—This was a severe attack, the pyrexia lasting for 25 days. There were rigors on the sixth, seventh, and eighth days of the disease.

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Pneumonia developed nine days after the first rigor, and phlebitis nine days later. This patient had had malaria before. The patient recovered.

CASE 3.—The patient had been ill for several weeks before his admission, and he died on the sixth day after admission. While in the hospital he had several rigors.

CASE 4.—This was a severe attack, the fever lasting for four weeks. Rigors occurred on the last day of pyrexia, and again on the fourth, seventh, and tenth days of convalescence. The patient recovered.

CASE 5.—This was a severe attack, lasting for 63 days. Several rigors occurred during the attack without definite cause. The patient recovered.

CASE 6.—This was a moderately severe attack, lasting for 27 days. Several rigors occurred nine days after phlebitis had developed. Nine days later the patient was attacked with otitis media. The patient recovered.

CASE 7.—Rigors occurred during an ordinary attack without any definite cause. The patient recovered.

CASE 8.—The patient died from fever at the end of 21 days. Several rigors occurred during the attack.

CASE 9.—This was a mild attack, the fever lasting about 11 days. There were rigors on the last two days of pyrexia. Doubtful malaria parasites were found in the blood. The patient recovered.

CASE 10.—This was a severe attack, the pyrexia lasting for 59 days. The patient had had several rigors, and malaria parasites were found in the blood. He had suffered previously from malaria on the West Coast of Africa.

CASE 11.—The patient had been ill for some weeks before admission into the hospital, where he suffered from pyrexia for 18 days. Malaria parasites were found in the blood, and it gave the serum reaction. The patient recovered.

Arthritis.—There were eight cases of arthritis, an incidence of 3·05 per cent. In one case there was effusion. The following is a short account of these eight cases :—

CASE 1.—On the third day of convalescence there was pain in the fingers, shoulders, and knees, without effusion and without rise of temperature. Similar pain recurred on the nineteenth day of convalescence.

CASE 2.—There was pain in the hips and ankles occurring on the ninth day of convalescence ; this subsided in a few days.

CASE 3.—This was a severe attack of enteric fever with petechiæ and spongy gums, followed by a relapse. Pain occurred in the right elbow with effusion and redness. The temperature went up to 104·2° F. and there was slight fever for the next two days. The inflammation subsided in about a week.

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CASE 4.—On the fourteenth day of convalescence pain occurred in the left shoulder and arm, with slight pyrexia lasting for two or three days.

CASE 5.—On the twelfth day of convalescence there was pain in the right arm, hand, and ankle, the temperature being 103° F. Pyrexia lasted for about six days. There was a similar attack a month later which lasted for seven days.

CASE 6.—On the last few days of the pyrexial stage pains occurred in the knees and elbows, lasting for several days.

CASE 7.—During the pyrexial stage pains occurred in the back, shoulder, and ankle, and lasted for two or three days. This patient had had rheumatic fever before.

CASE 8.—During convalescence rheumatic pains occurred in the right arm. This patient had previously suffered from rheumatic fever.

It will be observed that in the last two cases the patients had previously suffered from rheumatic fever, which was doubtless the cause of the arthritis. But in the others no other cause than the enteric fever could be ascertained. Most of them occurred during early convalescence. The arthritis may be compared to that arising in scarlet fever—a comparison which is supported by the occurrence of suppurative arthritis in a case of which we have only the notes of the *post-mortem* examination.

Epistaxis.—Epistaxis was very frequent during the early stage.

Tender toes.—There were a good many cases of tender toes, such as have been described by Dr. Handford. The toes were excessively tender, requiring a cradle to keep off the pressure of the bed-clothes. There was no swelling, and they all recovered.

Conclusions.—The conclusions which we draw from the analysis of these cases is that the type of typhoid fever met with in South Africa does not differ in any essential respects from that met with in England and America. The mortality and the incidence of complications are much the same as in the variations which are met with elsewhere. The only complication which appears to be specially prominent is phlebitis, which occurred in 5·6 per cent. of our cases. This was not very much higher than in Dr. Poole's cases at Maidstone, and in Dr. Caiger and Dr. Goodall's cases at the Metropolitan Asylums Board Hospitals, which were 3·8 per cent. and 3·4 per cent. respectively. We do not feel that inoculation has any marked influence either in preventing or in modifying the disease.

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Dr. WILLIAM CAYLEY said that, as he had not been out to the war, he knew nothing of it, except what he read in the papers. Therefore he was hardly competent to discuss, much less to criticise, the conclusions which had been come to by those who had the opportunity of seeing the disease in South Africa. The paper was interesting, as showing the identity of the epidemic with typhoid under very varying conditions and climates in different parts of the world. There were many interesting points in the causation and pathology of the disease which he hoped would receive some elucidation from the observations made at the seat of war. In England we were perhaps disposed to take a too one-sided view of the causation, due to the fact that in all the widespread epidemics at home the causation was traced to contaminated water, either directly or indirectly, and one was thus apt to overlook other causes. The water supply did not appear to be the only cause of the epidemic in South Africa, nor in those in India.

The statistics showed how extraordinarily contagious the disease was. Its spread in hot climates had been attributed to its dissemination in dust, and experimentally it had been shown that the bacillus could be dried and blown about, and then give cultures. Insects had been thought to be a cause, and experiments had been made to see whether the excreta of flies could be made to give cultivations. If flies conveyed the contagion, he thought it could hardly be so much by the excreta as by the feet.

The disease was communicated to animals by inoculation, but they did not have enteric fever, although they got a fatal blood-poisoning. The same had been observed in the human subject. It would be interesting to know whether any cases had been met with in South Africa in which the typhoid bacillus was present in cases of pneumonia and meningitis, for instance, without the patient suffering from actual enteric fever.

He thought Dr. Washbourn was to be congratulated on the very low mortality in his hospital, considering the unfavourable conditions to which the patients had been previously subjected.

With regard to the effects of inoculation, he, Dr. Cayley, had little personal experience. But he did not think the statistics which had been given that night were sufficient to invalidate those which showed more favourable results from inoculation on a larger scale, both in India and in South Africa, and even in this country. He hoped that when all the statistics from South Africa were available they would enable a definite conclusion on the question to be arrived at. He regarded the evidence in favour of the procedure as still sufficiently strong to justify or render imperative the inoculation of persons who were sure to be exposed to the infection.

Professor WRIGHT (Netley) complained that, in speaking of inoculation, Dr. Washbourn had not discriminated. To say that typhoid inoculation was of no use because it was not very successful under the special conditions which prevailed in South Africa was very much like saying that vaccination and antidiphtheritic inoculation were of no use, because under adverse circumstances they did not give brilliant results. Many things had to be taken into consideration, such as the element of time and the condition of the patients when they came to hospital. Because the statistics did not seem favourable under the difficult conditions in South Africa—and he could say they were indeed difficult—was it impossible to get good results from inoculation among people who were not exposed every day to typhoid infection, but who were living under good conditions

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and were fed properly? He understood Dr. Washbourn that because typhoid inoculation was held to have failed under the difficult conditions obtaining in South Africa, therefore if a young man came to him who was about to go to India he should advise him not to be inoculated. One might just as well form an opinion of antidiphtheritic serum, based upon its effect upon people in the last stage of diphtheria, and, in consequence, advise that children should not be inoculated. The matter required to be tested. Though he did not make the organism, nor yet the body, yet he was responsible for making the typhoid culture and for sending it out, and he had to see that it was not strong enough to kill people. If it was not successful in South Africa it did not follow that it would not be in India. There was also the question of the lapse of time. Dr. Washbourn had brought forward statistics, but in order to make them valuable it was necessary to know when the persons had been inoculated. He found that an excessive dose of the vaccine rendered the patient more susceptible, so that such an one, if exposed to typhoid soon after inoculation, would be infected more quickly. He had known of second successive attacks, and, in one case, of a third attack. They had only followed the clinicians in the inference that the more severe the reaction one could get into a man, short of killing him, the greater the protection would be. But Dr. Washbourn should have mentioned the period after inoculation at which the cases occurred. Typhoid inoculation, like vaccination, would not protect the person for his whole life. Again, Dr. Washbourn had not said whether those inoculated had one dose or two. He (Professor Wright) recommended that men should be inoculated twice. He knew the difficulties; he had a friend sitting beside him who was inoculated once, but felt so ill that he would not consent to a second dose. Dr. Cayley, in his hospital, had 62 people doubly inoculated, with no cases of enteric, though they were exposed to a great deal of infection. Knowing that Dr. Cayley's figures were not absolute in all cases, Dr. Washbourn had not included them. In this he was conscientious; but there was such a thing as over-conscientiousness.

With regard to Ladysmith, the statistics showed that 2 per cent. of the inoculated had enteric, and 14 per cent. of the uninoculated. Two inoculated officers died, it was asserted, of enteric, and he was plainly told that those two men would rise up and judge him. He was anxious to get at the history of those two cases, and found that one had not been inoculated with his serum at all, but with another's preparation; and the second case died of an ailment which was certainly not typhoid. He therefore had to delete those two. He knew that some of the figures were not exact, but by ignoring them there was a danger of doing an injustice to the higher truth. He took all the figures available—including Dr. Washbourn's and those at other hospitals in South Africa, and those in India—and he had stated elsewhere the difficulties under which he had to work—and adding all those figures together he got a total of 8,600 inoculated people and 41,000 uninoculated. He proposed to publish a synoptical table of the results. Some of the men were exposed every day, others led comparatively easy lives, but in all cases figures were compared of men living under similar conditions. The net result was that among the 8,600 inoculated there was a mortality of 2¼ per cent.; and among the 41,000 uninoculated, under the same conditions, 5¾ per cent. Therefore, taking the results from all the world, some being done under very difficult circumstances, and in some instances the serum not being properly made, there was a reduction of the cases of about 2 to 2½ per cent. as the result of inoculation.

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With regard to case mortality, the returns were obtained with very great difficulty, but worked out at 12 per cent. mortality among the inoculated, 20 per cent. among the uninoculated. He took his stand upon those figures, and was not advocating a nostrum of his own, but was dealing with common knowledge. To protect against a disease, it was necessary to give a dose of animal disease first. The great point was to find the right dose; and he thought he had found a method of doing that. But it would not save the whole world from typhoid. He now knew that in typhoid they were dealing with a disease which, in a great number of cases, did not give protection. Therefore the problem was more difficult as they advanced. But the hope of the future lay in inoculation; it was well known in the profession that no man could reduce the deaths by 75 or even 60 per cent. by any drug, or by means of anything but inoculation. But an enormous amount of actual clinical observation was necessary for proper advance to be made in the matter. He was deeply grateful to people who pointed out where he was wrong. Dr. Cayley's experience was that where inoculation had been done the attack was very slight. Dr. Washbourn had referred to the youth of some of the attendants, but did not say whether age made a difference. In Dr. Washbourn's statistics there was a reduction of incidence and a reduction of case mortality, and he would take care to add them to his statistics. When he compiled his synoptical table he thought it would turn out that there was a certain advantage in inoculation, and the advantage would show most strongly where people were least tried. It was our only hope in dealing with the scourge of enteric fever.

Surgeon-General Henry Cayley said he had figures in connection with the subject which were certain; they were not open to the doubt attendant upon information based upon what the patient said. A large number of patients were not in a mental condition to remember whether they were inoculated or not. Some would wilfully deceive because they were afraid that a negative reply would result in inoculation. Others did not know whether they were inoculated or vaccinated. In some ships inoculation for enteric was done in the arm, and then Tommy did not know whether it was inoculation or vaccination.

The staff in the first section of the Scottish Hospital numbered 61, seven of them being nurses. Of the remaining 54, all but three were inoculated twice. Of those three, two were inoculated once, and the other, a sergeant-major over 50 years of age, refused to be so treated. Of those 54, not a single one had enteric, although they were exposed more, and for a longer time, than those in the second section, many of them having been at work at Bloemfontein in the very height of the epidemic there. Of the number (54), about 25 were medical students, aged 19 to 23, which was generally considered the most susceptible age for enteric fever. In the second and third sections there were 102 people, 28 of them being nurses, with whom he would deal separately. The majority of the remaining 74 were inoculated twice, but a smaller number of them only once, and a few not at all. Of the 74, five got enteric, of whom three had not been inoculated, and two had been inoculated once. Of the three who had not been inoculated, one died, and of the two inoculated once, one died. He found on inquiry that when that man was inoculated there was no reaction, which seemed to show that that inoculation was imperfect. Thirty-five nurses in all went out in the different sections of the hospital, and 32 of them were inoculated twice. Two were not done

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because they had had enteric a year or two before going out, and it was not therefore thought to be necessary. One refused to be inoculated, and she was the only nurse who had the disease there, her attack being very severe. Those figures strongly supported the value of inoculation for a time; he could not say for how long, but certainly for five or six months. With regard to ordinary patients treated in the hospital, the facts were not so reliable: of 92 admissions for enteric there were 11 deaths; of the 92 cases, 15 were said to have been inoculated, a few were doubtful, and about 70 non-inoculated. Of the 11 deaths, one had been inoculated once, and 10 not inoculated. His own impressions were that the disease was less severe in the inoculated cases. He wished to suggest to Dr. Washbourn whether the cases in which there were symptoms of arthritis were cases of Malta or Mediterranean fever, as he saw several cases of this fever in South Africa which were diagnosed as enteric.

With regard to the inoculation of the staff, Dr. Dodgson examined the blood of a considerable number of the staff. Of 23 people of the first section he found a high reaction in every case except two, and it was found that those were the only two who had been inoculated only once. Of the second set which went out, Dr. Dodgson examined the blood of 22; they were not selected cases, but men who were off duty at the time and available. In 11 there was no reaction, although they had been done a month later than those in the first section. In nine there was a faint reaction, and in two a full reaction, which seemed to show that the inoculations in the second section had not been so complete as in the first.

Dr. H. H. Tooth said Dr. Washbourn's figures had been somewhat of a surprise to him. As he had shown in his paper before the Clinical Society, his experience had been in favour of inoculation. Of course the numbers dealt with were very small, but such as they were they seemed to be significant.

His experience had led him to think that the severer the constitutional reaction to inoculation, the lower the degree of immunity, so that such a person should be subjected to one or more re-inoculations. Unfortunately a man who has suffered much at his first inoculation is likely to be very shy of subjecting himself to such an experience again. He was, therefore, glad to hear that Professor Wright had suggested a diminished dosage, which, repeated once or twice, would produce the same ultimate results in a less painful manner.

The question seemed to him to be one of immunity, which varies with the individual. This may be so high that a man may go through many campaigns subject to infection without getting the disease. In others this immunity may be so low that even a previous attack may not offer protection, and in these inoculation could not be expected to be of much use. But he thought that there was a large intermediate class in which inoculation might raise the individual immunity so as to diminish the liability to infection, or to lower the mortality.

Dr. Rolleston expressed his high appreciation of Drs. Washbourn and Elliot's paper. He had succeeded Dr. Washbourn at the Imperial Yeomanry Hospital, Pretoria, and therefore many of the cases making up the speaker's statistics which had been quoted by the readers of the paper had been actually under Dr. Washbourn. With regard to inoculation—to which the discussion had been almost exclusively directed—his figures had been already quoted, and they were small. So far as they went they showed that in South Africa inoculation did not absolutely protect against

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enteric fever. In fact, there might even be a greater incidence of enteric fever among the inoculated than among the non-inoculated.

Possibly this was an accident, but it actually occurred among the *personnel* of the Imperial Yeomanry Hospital; among 150 persons (medical officers, nurses, and orderlies) there were 22 cases of enteric fever, or an incidence of 14·6 per cent. Of the 150, 35 were inoculated, and of these, six, or 17 per cent., contracted enteric, while of the remaining 115 non-inoculated members of the *personnel*, 16, or 13·9 per cent., suffered from enteric fever. As shown by statistics, there was some reason to think that inoculation protected the individual from contracting enteric fever for six months or more; in 19 officers who contracted enteric fever after inoculation, the interval between inoculation and the fever varied between one and 21 months, but on an average was 30 weeks. Further, his figures, though small, seemed to show that inoculation protected against a fatal termination. Too much stress should not be laid on this point, for after all, death in typhoid fever might be due to some secondary affection or to an accident, such as hæmorrhage or perforation, and was not always proof of the severity of the typhoid infection *per se*. He thought that some features of enteric in South Africa, such as the frequency of second attacks and relapses, could be easily explained by Mr. H. E. Durham's* new theory of relapses in fever, and, if this were so, it would be well to make the vaccine of dead typhoid cultures as polyvalent as possible, and to obtain the bacilli from many different sources, and especially from South Africa. To turn to one or two other points, he was inclined to think it well to distinguish simple pain in the joints (arthralgia) from pain accompanied by effusion (arthritis). Post-typhoid or typhoid pleurisy was commoner on the right side of the chest. As to the cause of phlebitis, which every one agreed had been the commonest sequela of enteric fever, he was inclined to refer it to diminished resistance of the veins of the legs brought about by the fatigue of "trekking." It would be interesting to know the extent of the thrombosis in these cases; it was a most noticeable fact that pulmonary embolism from phlebothrombosis, after enteric, seemed to have been unknown in South Africa. A last point of interest was that patients had been admitted for dysentery and, while convalescent, had manifested the symptoms of enteric fever. In many of these cases the facts were quite compatible with the view that the dysentery and enteric had been contracted at the same time, possibly at the same draught of foul water.

Surgeon-General H. S. Muir (having been invited by the President to make a few remarks) said he thought the question of inoculation largely resolved itself into one of numbers: 100, 200, or even 500, were of very little use for basing a conclusion. Thousands of cases were required, both from the seat of war and from India. He was very anxious to learn the net result. He expected to hear more in the debate about causation, a point Dr. Washbourn did not touch upon. He did not think water was the only source of contamination, though no doubt it was a very important one. In the same way as one might take a horse to the water but could not make him drink, so Thomas Atkins would not drink pure water when impure water was handier for him.

Dr. E. W. Goodall thought too much stress had been laid in this country upon the influence of the water supply in the causation of

* Durham, 'Journ. Path. and Bacteriol.,' vol. vii, p. 240.

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enteric fever. A mistake had been made in teaching that typhoid fever was not infectious. Murchison's dictum, that those who attended on typhoid patients never contracted the disease, was quite wrong. Nurses and attendants in fever hospitals often contracted it, though patients admitted with other diseases did not. The South African campaign had shown that water was not the only agency in the spread of typhoid fever. In the various papers which had been published recently about typhoid fever in South Africa, one was struck with the similarity of the disease to that seen in England. Arthritis as a complication was very rare; he had only seen two undoubted cases of it which were not rheumatic in any sense. He had once seen ulcerative colitis following typhoid fever.

With regard to inoculation, he had been favourably impressed with its value, having regard to the figures given in several recent publications. But there were many points which required elucidation. Professor Wright had recently published a very interesting paper on the subject in the 'Lancet,' in which he said that in order to protect people they should have a small dose of the vaccine, so as to produce a slight reaction. He (Dr. Goodall) understood that previously doses had been given large enough to produce a well-marked and often severe reaction; at any rate, the reaction seemed to have been well marked, for people had objected to having a second dose. But he now understood Professor Wright to say that a large dose of vaccine would make a person more susceptible to typhoid fever. It was claimed that those who were inoculated before did not get the disease, or only in smaller numbers; yet one would have thought that having had large doses of vaccine they would have contracted the disease in larger numbers than the uninoculated, according to Professor Wright's latest ideas. Professor Wright claimed a reduction of 2½ per cent. of incidence in some thousands of cases he quoted, but he (Dr. Goodall) did not think that so small a difference was of much account. The ages of those inoculated and not inoculated should be given in five yearly groups. Professor Wright had published a paper in last week's 'Journal,' concerning an outbreak of typhoid fever in a Dublin asylum, but more information was wanted in regard to that. It was a question between the staff and the ordinary inmates. The staff had not been inoculated, and they got the disease. But it should be stated in more detail than was stated in the paper whether the staff were exposed in the same way as the patients were. Again, a very important point was the age. Among the inmates of some asylums there was a preponderance of old people, who do not contract the disease so readily as the young. Professor Wright was probably correct in his conclusions, but his case would be strengthened if he were to bring forward those data.

Professor WRIGHT, in answer to the last speaker, said that in his article on the outbreak in Richmond Asylum, Dublin, he specially mentioned that persons above 55 years of age were excluded from his figures. He also there stated that the nurses and attendants were exposed to the same degree as the patients; they lived in the wards where the patients were kept.

With regard to his other contribution on the subject, he was dealing there with the incubation period of healthy people whom he was inoculating himself. After a big dose there was a susceptible period, but not after small doses. He tested his own blood a few days ago, and found it in a very healthy condition, and he was not sure he had not

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benefited by giving himself a big dose. The other men had gone to India, and he could not examine their blood. They had a smaller dose than he had, and though their blood gave better results in a week, yet he fancied that his would be best at the end of a longer time. If the clinical symptoms were disagreeable, a smaller dose could be taken, and after the establishment of that degree of immunity, a larger one.

Dr. WASHBOURN, in replying on the discussion, said he regretted he knew nothing about the contraction of typhoid fever in other ways than through the intestinal canal. They knew nothing of enteric fever which showed itself under the guise of pneumonia, but it was a point which it would be most important to establish.

With regard to inoculation, Professor Wright had raised many questions, but he thought that gentleman had misunderstood the paper, which did not state that the procedure was of no use under any circumstances. The authors referred to South Africa, and especially to their own cases. It was true that inoculation was put to a very severe test in South Africa, and that generalisations based upon such a test were hardly fair. But it was just under those circumstances that inoculation would be of value. Even if inoculation were proved to possess marked protective powers, he did not think it would be much used in England, as the exposure to infection was not great, and the constitutional disturbances produced by inoculation were severe. He knew of a case of a medical officer who was ill for three weeks with pyrexia immediately following inoculation.

Professor Wright gave some interesting experiments with regard to the effect of a large inoculation in increasing temporarily the susceptibility to enteric fever. He (Dr. Washbourn) did not know upon what that statement was based. As far as he understood, it was simply upon the bactericidal properties of the blood.

Professor WRIGHT said it was partly that; a guinea-pig in that condition succumbed to a small amount. In human beings they could only go by the blood examination.

Dr. WASHBOURN, continuing, said he did not think it was definitely proved that susceptibility to typhoid fever was in direct relationship to the bactericidal quality of the blood.

He thought there was definite proof that age had a very marked bearing upon susceptibility to typhoid, younger people being more prone to it, as was shown by the Metropolitan Asylums Board's statistics. He might say that the dressers who caught the disease at Deelfontein were younger than the rest of the staff. Among the 10 inoculated ward-maids one contracted enteric. The mortality among the patients who had been inoculated was higher than among those who had not been inoculated—the mortality being 16 per cent. and 10·7 per cent. respectively.

With regard to patients not knowing whether they had been vaccinated or inoculated, the expedient adopted was to make inquiries as to the procedure and the subsequent history, which prevented any error on that score. The reason Professor Cayley's cases were omitted from the statistics was that in some cases he gave numbers, and said "nearly all of whom were inoculated."

With regard to arthritis, Professor Cayley asked whether any of the cases mentioned were really Malta fever. There were none included in

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these cases, though he had seen and recorded cases of the disease in South Africa. The course of Malta fever was much longer than that of typhoid. The arthritis in enteric was of a transient character, and was comparable to that of scarlet fever. In one of the cases there was serous effusion, and in another pus in the joint.

With regard to the theory of relapses quoted by Dr. Rolleston, he did not think there were any facts to support it. Nothing was known with regard to any difference in the agglutinative power of the blood during the primary attack and the relapse, as far as its specific action on the different strains of bacilli was concerned.

With regard to the suggestion that phlebitis was due to strain on the legs during marching, most of the cases in their paper were among mounted men, but that did not show very much, because they did not know the proportion of mounted to unmounted men in hospital. He thought the frequency of phlebitis was due to the absence of salts in the food, especially vegetable salts.

He agreed that enteric could be, and often was, conveyed by means other than by water—by flies, dust, and by direct infection.

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November 25th, 1901.

THE SYMPTOMS AND TREATMENT OF MOVABLE KIDNEY.

By HENRY MORRIS, M.A., M.B. Lond., F.R.C.S. Eng.

GENTLEMEN,—When asked by the medical secretary to open a discussion on movable kidney my natural antipathy to take part in controversy disposed me to decline, but I consented on reflecting that the subject, though none of my choosing, is one upon the due and proper comprehension of which it is worth while to spend time and trouble.

I propose, in the remarks which I am about to make, to state in the first place what I understand by the term "movable kidney"; secondly, to explain the great discrepancy of opinion as to the frequency of movable kidney; thirdly, to describe how this mobility may be ascertained to exist; fourthly, to allude very briefly to the symptoms, diagnosis, and effects of movable kidney; and lastly, to indicate the class of patients benefited by operative treatment.

WHAT IS TO BE UNDERSTOOD BY MOVABLE KIDNEY?

Contrary to the inferences to be drawn from some of the many works on the subject, a certain degree of mobility is natural to the kidney. Surgeons accustomed to operate upon this organ frequently witness when the kidney is exposed in the living body that it has a limited movement in a vertical direction, descending with inspiration and ascending with expiration. Deep inspiration and forced expiration increase these movements. This slight natural descent of the kidney in inspiration cannot be detected by palpation in every individual; in some persons the organ does not descend sufficiently to enable even its lowest pole to be felt on the deepest inspiration. This, however, is not tantamount to saying that every kidney which descends during inspiration, so that the smallest part of it can be felt, is a kidney the means of fixation of which are insufficient—that it is, in fact, a movable kidney. Many cases in which the lower pole of the kidney, though beyond reach during ordinary respiration, yet comes within range of palpation during a

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full inspiration, and returns on quiet expiration, are uncomplicated by symptoms, and the range of their movement is simply natural. But a kidney is deficient in its proper means of fixity and must be considered abnormally movable under any of the following conditions :—

1. When the whole kidney descends during deep inspiration below the examiner's fingers, on bimanual palpation.

2. When the whole or the greater part of the kidney so descends that it can be *felt* between the fingers of the two hands.

3. When the lower half of the organ so descends and can be so felt. In the cases in the third group and in some of those in the second group, the organ slips back again in expiration and cannot be *retained* between the fingers; but in many of the cases in the second group and all of those in the first group, the kidney can be retained between the fingers of the two hands, even after the patient has taken a full expiration. These three groups of movable kidney, together with the cases above referred to, in which the kidney can be felt no matter to how slight a degree, on the deepest inspiration, are described by Glénard as "movable kidney of the hypochondrium," or the "new form of movable kidney" (*rein mobile nouveau*).

4. There is a group of cases in which the kidney is often out of position during natural respiration; and in some extreme cases in this group the organ may even be seen at a glance projecting the abdominal wall, or can be felt at once on simply placing a hand upon the front of the abdomen. This group includes the so-called "floating kidney." This is the group which Glénard calls "the movable kidney of the flank and of the iliac fossa," or "the classical movable kidney." If I understand him correctly, he actually believes that these last were the only cases known to or recognised by the profession prior to 1886, and he claims to have himself discovered all those varieties which he includes under the term, "the new movable kidney" (*rein mobile nouveau*) by the employment of his own method of bimanual palpation—namely, the "*palpation néphroleptique*" as he calls it. But certainly for the last 20 years at least some of us in this country have been employing a method of palpation of the hypochondrium very similar to his own, and have recognised some of the forms of movable kidney which he calls *new*, and of which he claims to have been the discoverer.

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5. Besides the varieties of movable kidney above enumerated, I have been in the habit of describing another, and by no means the least important. These are the cases in which the kidney moves in a "cinder-sifting" manner behind the peritoneum, where the organ slides, so to speak, upon the plane of the posterior parietes, never "dropping" forwards or inwards, and so not properly described by the term "nephroptosis." Moreover, the kidney sometimes rotates more or less round transverse axes, or round a vertical axis. When round the transverse axis or axes one of the poles of the organ (more usually the lower pole) glides inwards, or swings forwards and inwards, and this displacement may be combined with movements in the vertical or lateral direction. In vertical rotation the convex or outer border advances.

How the Discrepancy of Opinion as to Frequency of Movable Kidney can be Explained.

Though no one would, I imagine, in the present day repeat the statement formerly made that there is no such thing as a movable kidney, and that all the instances described had been cases of mistaken diagnosis, still there exist the greatest differences of opinion as to the frequency with which the kidney can be found to move pathologically. All sorts of variations between 1 and 22 per cent. and between 3 and 46 per cent. have been given as the frequency of movable kidney in the female sex. Glénard, from his experience as physician to the Thermal Hospital at Vichy, met with 537 cases in 4,215 patients under his observation, or a proportion of 14 per cent. of all the patients, males and females, examined, and 22 per cent. of all the females. Senator and Guttmann,* on the other hand, allow of only from 1 to 3 per cent., and the conclusions drawn from the reports of *post-mortem* examinations yield in some of the statistical tables less than 1 in 1,000. These great discrepancies are capable of being easily explained.

1. Many cases have been overlooked in the dead body, the inspections having been made without any special examination as to the existence or otherwise of undue mobility, only the very striking and extreme cases which could hardly fail to attract notice on abdominal inspection being notified.

* 'Berliner Klinische Wochenschrift,' 1890, No. 15, Discussion.

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2. In the living body many cases are daily overlooked, because the old teaching that the kidney does not come under the influence of respiratory movements has prevented the employment of those methods of examination by palpation which reveal renal mobility, and also because the method of bimanual palpation commonly employed, and which is confined to manipulations below the ribs, generally checks or prevents the respiratory movements of the kidney by keeping or crowding the kidney back into the renal fossa.

3. Those who deny that the kidney can be detected by palpation during normal or even deep respiratory movements, unless the organ is itself abnormally mobile, include in the category of movable kidney every case in which during deep inspiration the lower extremity only of the kidney comes within reach of the examiner's fingers.

4. Physicians at health resorts and thermal hospitals, the greater number of whose patients are dyspeptic, neurotic, or hysterical, if they systematically employ methods of palpation with the object of detecting the existence or otherwise of abdominal ptoses, undoubtedly find a greater percentage of movable kidneys in the persons whom they examine, than other practitioners who have fewer patients suffering from such-like maladies and who do not systematically examine for visceroptoses; and this percentage will be made still greater if, like Glénard, these physicians include every case in which they can feel the lower pole of the kidney on deep inspiration as one of movable kidney or nephroptosis.

How to Ascertain whether the Kidney is Movable or Not.

Several methods, each effective, have been recommended, and a brief description of two or three of them is desirable here because none of them seem to be as generally known or employed in clinical practice as they ought to be. The method which I have myself used since I first directed my attention to the surgery of the kidney, is the following:—The patient should be on his or her back with the shoulders slightly raised by a firm pillow, the spine should rest quite flat upon the sofa or mattress with the lower limbs very slightly flexed. The patient should be lying close to the right edge of the bed or couch for the examination of the right kidney, and

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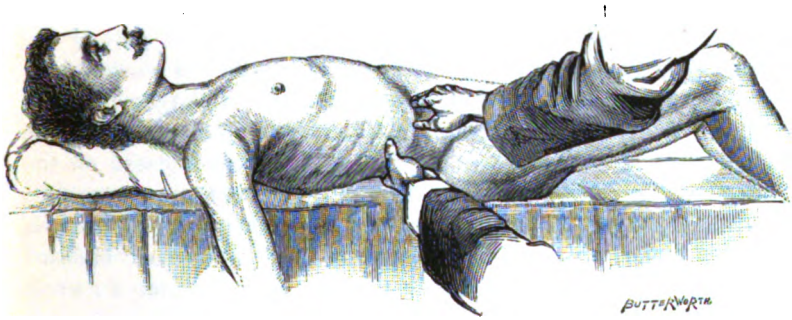
close to the left edge of the bed for the examination of the left kidney. The surgeon then places the fingers of one hand behind and upon, and also just below, the last two ribs, and the fingers of the other hand flat upon the front of the abdomen, just below the costal margin near the outer border of the rectus muscle. (*See figure.*) He should be on the same side of the patient as the kidney which he is about to examine, and should be sitting or kneeling at the bedside, so as to have his lowermost forearm as nearly as possible level with the patient's body. When examining the right kidney the left hand should be on the loin and the right in front; when examining the left kidney the right hand should be on the loin, and the left on the front of the abdomen. With the hand on the loin the lumbar parietes should be gently but firmly pressed forwards,



Showing position of patient and examiner's hands.

and sustained so; then the fingers of the hand in front should gently and steadily depress the anterior abdominal walls upwards, backwards, and outwards. The kidney, if enlarged or displaced at all, will now be felt between the two hands, and even if not enlarged the lower pole of the organ will, in some persons, be detected by the fingers during natural respiration. Should this not be the case the patient should be directed to take a long and deep inspiration, and suddenly, but without muscular effort, to allow the air to escape from his lungs; in other words, he should make a long-drawn "sigh." The hand behind still maintains forward pressure on the loin, and the hand in front quickly follows the receding abdominal wall as it becomes relaxed in the expiratory movement. Thus the kidney, which otherwise cannot normally be felt, can often be seized between the fingers of the two hands before it has time to gain its

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normal position. This method becomes even more efficacious if the patient takes a second and a third similar "sigh" whilst the surgeon maintains and increases the deep pressure made after the first and second. Palpation is sometimes assisted by raising the patient's shoulders so that he assumes the semi-recumbent attitude.

In testing the mobility of the kidney, it is sometimes advantageous to place the patient in the knee-elbow or in the upright position. As a rule, however, I find it best to get the patient to roll over on the opposite side and then to employ bimanual palpation as I have just described. A movable kidney which persistently declines to become evident will frequently fall at once towards the front of the vertical column, below the costal margin, as soon as the lateral position is assumed and a deep inspiration is taken.

Glénard's method consists in compressing the lower part of the thorax by placing the fingers of one hand on the eleventh rib posteriorly and the thumb of the same hand on the level of and below the extremity of the ninth rib, then crowding back at the same time the abdominal walls with the other hand placed on the umbilical region. By compressing the intervening structure between the fingers of the two hands, the kidney, as it descends during a deep inspiration, is detected by the thumb. Glénard describes the thumb and fingers as assisting in forming a narrow ring, which is completed on its internal side posteriorly by the vertebral column, and anteriorly by the fingers of the other hand. He describes the kidney as feeling like a vague indefinite mass as it descends during a deep inspiration. This is the "*palpation néphroleptique*" of Glénard.

Israel's method consists in making the patient lie on the side opposite to that which is to be examined, whilst the surgeon stands on the left side of the couch to examine the right kidney, and on the right side of the couch to examine the left kidney; in each case, therefore, his own face is towards the patient's. In examining the right kidney he places the fingers of the left hand upon the right lumbar region and the right hand upon the anterior abdominal wall in such a manner that the tips of the index finger and the middle finger are two fingers' breadth below the junction of the ninth and tenth costal cartilages. Then, whilst the left hand is supporting the lumbar region, the patient takes a deep inspiration and the kidney is felt at the end of this act.

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By my own method the kidney is felt best at the end of the inspiration, and at the very beginning of expiration, as soon, that is, as the muscles of the abdominal wall relax. Glénard claims for his method that it is at the beginning and throughout the act of inspiration that he feels the kidney. But a result of making a deep inspiration is to push forwards and to harden the muscles of the anterior abdominal walls, and this has the effect of preventing deep indentation of the parietes with the hand which is in front. It is only in very thin persons, and in those with very flaccid abdominal muscles, or in very extreme cases of nephroptosis, that the kidney can be felt to descend during the whole act of inspiration. By Israel's method the detection of the kidney is made at the end of inspiration.

The Symptoms of Movable Kidney.

These may be considered under the headings of, first, the physical signs discoverable by palpation; and secondly, the subjective symptoms complained of by the patient.

The Physical Signs.—There may be none ascertainable, owing either to the thickset conformation or the obesity of the patient; or, in a slight person, to the elongated shape of the thorax, to the high position of the kidney, or to the movements of the kidney being of the kind which I have described under group 5 as of the "cinder-sifting" variety. In thickset persons, and those inclined to stoutness, there may be a wide range of movement, and yet the kidney may be quite beyond the reach of palpation. Yet in some of the cases in which there are no physical signs the subjective symptoms are very acute.

In some cases of great mobility in which the kidney can often be detected very easily, there are times when the organ cannot be felt at all. I think it important to observe the rule never to declare that a kidney is not movable because on a *single examination*, even though most carefully and systematically made, I cannot feel it move; more especially not to do so if a trustworthy observer has on some previous occasion felt sure of its moving. I have sometimes reduced a floating kidney, and nothing that I or the patient could do at the time of the consultation could make it come again into evidence, though the next day it was floating as freely as ever. I once examined a patient in my consulting room who had been sent from Switzerland to have an operation for movable

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kidney, but I could find no evidence at all by palpation that the organ moved. Two days afterwards I saw the patient in her bed, when the right kidney was found to be so movable that it could be pushed freely about in almost any direction. There is no doubt of the importance of making more than one examination where movable kidney is suspected.

When the mobility is considerable, the kidney may fall below the margin of the ribs towards the umbilicus or the pelvis when the sitting or erect posture is assumed, or it may roll towards the median line when the body is inclined to the opposite side, and be easily pushed back into the loin when the recumbent position is resumed. In rare cases the patient can at will bring her movable kidney right out of the hypochondrium into the umbilical, lumbar, or iliac region, by some particular movement or posture which she has discovered by accident. Thus a middle-aged woman with flaccid abdominal walls was able with her right hand to push out the right kidney without any pain, so that it could easily be grasped through the flaccid abdominal walls by the fingers of one hand.

Except in the "cinder-sifting" form, one can make out by palpation, not only if the kidney moves too freely, but also if it rotates. The direction of the long axis of the organ is frequently oblique from above downwards and inwards, the hilum being turned upwards and inwards. More rarely the hilum is turned downwards. When the patient is rolled over on to the sound side, the convex border of the kidney may be felt to tilt forwards and to incline inwards towards the front of the spine.

When the kidney is ectopic, occupying the lumbar or iliac region—whether it be only of normal size or enlarged from some disease—it is generally beyond the influence of the diaphragm, and does not move with respiratory movements, though it may be freely pushed from place to place.

Percussion over the kidney usually gives a tympanitic or muffled tympanitic note. Epigastric aortic pulsation and congested enlargement of the movable kidney are very commonly present. During the catamenial period I have known a movable kidney to assume large proportions, and to have been mistaken for a new growth of the kidney.

The Subjective Symptoms.—The kidney may be distinctly movable without any symptoms whatever being produced, and without the

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patient being at all aware of its undue mobility. The usual symptoms are (1) pain, (2) troubles of digestion, and (3) neurasthenia and hysteria. These three kinds of symptoms may occur either separately or in association. (4) Occasionally, but comparatively rarely, there is some change in the urine or in the functions of the urinary organs. (5) More rarely still, some extreme complication due to compression or dragging, such as intestinal obstruction, jaundice, or great gastric dilatation occurs.

The pain is sometimes described as being merely a constant ache in the back and down the affected side of the abdomen, or as a dragging sensation or weight in the loins and on the affected side of the abdomen, or pain below the shoulders and at the back; or stiffness of the back and cramping pains in the abdomen may be complained of; and some patients experience an occasional uncomfortable sense of something moving in the side, which when very marked has been compared to the movements of a fœtus in utero. The pain may disappear on lying down, and become aggravated, if not actually induced, by walking and standing; in other cases it gradually increases, and remains severe even for hours after the recumbent posture is resumed. Neuralgic pains in the course of the great nerve trunks on the affected side may occur. Renal paroxysms of grave severity are in some cases excited. These attacks are quite sudden, and are characterised by intense abdominal pain, hardening of the abdominal wall uniformly or only on the affected side, faintness, giddiness, and symptoms of collapse. During these attacks the kidney can often be felt enlarged from congestion or renal retention of urine, and it is extremely tender. The urine becomes scanty, and may contain albumen, casts, or even blood; but again becomes normal when the attack passes off, and then polyuria sometimes follows. These symptoms are probably due to displacement and rotation of the kidney, with torsion of its pedicle, and to the organ becoming intensely swollen and congested. Acute hydronephrosis may be produced and a tumour thus formed, or a very tense hydronephrosis may occur in a kidney too small to form an appreciable tumour. Acute, paroxysmal, and severely painful cases have long been known, and have received the name of "renal incarceration," from their analogy to cases of strangulated hernia. I made mention of them in my work in 1885, and I published instances of the kind many years ago, and again in my Cavendish Lecture in 1893. Mr. W. Bruce Clarke, who recorded

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some cases in 1893, proposed for them the name "acute renal dislocation." This term had also been used by the ancient writers, who looked upon the condition as being more allied to the displacement of the head of a bone than to a strangulated hernia.

Disturbances of the digestive system are in some patients especially prominent. Vomiting, often frequent and severe, sometimes preceded by a burning sensation in the epigastrium, together with symptoms of gastritis or actual dilatation of the stomach, occurs. In the large intestine the most common condition is one of obstinate constipation, which may, in some cases, amount to temporary obstruction; occasionally there is irregularity of the bowels or diarrhœa. These symptoms are probably produced either by actual pressure or by the dragging of the misplaced kidney upon the intestine. Folds of peritoneum have been observed passing from the kidney to the duodenum in such a way that, when they are pulled upon, the lumen of the duodenum is narrowed or even occluded, as in a case recorded by Franks. Macalister, who has also observed similar folds, believes that they are by no means uncommon, and that they furnish a natural and adequate explanation of the gastric disturbances which occur in these cases. The gastric symptoms are liable to acute exacerbations or "crises," as first described by Dietl in 1864. They consist of violent attacks of colic attended by nausea and vomiting, and these may be accompanied by abdominal extension and tenderness, and sometimes by a rise of temperature and signs of collapse. Transient attacks of jaundice are said not to be infrequent, but I have never personally witnessed them; they are probably dependent in some way upon the dragging of folds of peritoneum on the duodenum as above described; for the idea that there is ever direct pressure of the kidney upon the bile-passages is not borne out by anatomical investigation, except possibly in such a case as Lindner's, in which a floating kidney with stones in the pelvis pushed against the gall-bladder and gave the appearance of a tumour of the gall-bladder, but in that case there was no jaundice. A right floating kidney, if carefully drawn forwards or downwards, causes traction on the duodenum at a point from two to three centimetres below the entrance of the biliary duct; and though it does not occlude the lumen, it may interfere with the contents of the bowel sufficiently to impair digestion and to cause biliary obstruction. Symptoms of chronic appendicitis are considered by Edebohls to be a very

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frequent complication of movable kidney on the right side, and he explains this on the supposition that the misplaced organ produces an indirect pressure upon the superior mesenteric vessels, which become compressed between the pancreas and the bodies of the vertebræ, and thus a chronic congestion of the appendix and colon is produced which is sufficient in the case of the appendix to give rise to definite symptoms.

As to neurasthenia and hysteria there is no doubt that movable kidney, like many other causes of physical suffering, slight as well as severe, may lead to the development of hysteria in a female who had previously shown no signs of any such neuropathic tendency, and this may be completely cured by nephropexy. There are other individuals who have shown signs of hysteria before the kidney has been known to be movable and whose condition is aggravated by the knowledge. But hysteria and neurosis are not by any means invariable accompaniments of movable kidney. Some of the most painful cases develop after a slight injury, and still many more, that occur without any assignable cause, are met with in healthy women, physically and mentally active, who are completely invalided against their will by the pain and digestive troubles caused by the movable kidney. Though the patients have perseveringly, but in vain, tried belts, diet, and recumbency for a long period, they obtain the most complete and gratifying results from fixation by operation. It undoubtedly happens sometimes that a person is credited with being neurotic and her symptoms are attributed to hysteria, because, owing to an insufficient investigation of the case, a movable kidney has been overlooked; but the opposite error of overlooking the hysterical element and attributing all importance to the nephroptosis is also made. To do this is to court failure, for we are regarding as a cause what may be only a coincidence.

As regards changes in the urine and the urinary functions: in most uncomplicated cases the urine is healthy, and is, as a rule, voided in the normal way; in many cases there is some albuminuria from time to time, but if the urine is habitually albuminous the albumen will be due to some pre-existing or co-existing disease. Still, there are cases, not so very few, in which urinary symptoms are complained of. Kuttner speaks of frequent straining in order to urinate, and of slight polyuria. Apolant draws special attention, in the case of a woman, aged 50 years, with very serious abdominal

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disturbance due to a floating kidney, to a symptom which had been but little regarded, if not entirely overlooked—namely, excessive polyuria. I have operated upon several cases of movable kidney in which slight pyuria or hæmaturia and frequent ardent desire to micturate were symptoms.

The leading question about which the most opposite opinions are entertained is as to the influence exercised by a mobile, an ectopic, or prolapsed kidney in producing any of the gastro-intestinal and nervous symptoms ascribed to it. It is said by Glénard and those who adopt his views, that nephroptosis is merely an accidental or casual incident in the progress of enteroptosis; that in this progress it is the ptosis of the intestines which is the essential element; that ptosis of the kidney, if the organ is otherwise normal, occurs only in nervous dyspeptics and persons affected with neuropathies or the affections grouped under the terms "biliary lithiasis" or "gastric crises," and that the fundamental symptoms in these patients are digestive; that the local subjective symptoms (the sensation of a ball in the side, the movements, the dragging from the loin, &c.), and the painful gastric and abdominal crises, are met with equally in persons whose kidneys are not mobile or displaced; and that these symptoms are of cæcal, colic, duodenal, gastric, or hepatic origin. Lastly, they aver that if a movable kidney of itself provokes any symptoms whatever it is because the kidney is diseased, and that then the symptoms differ from those commonly but erroneously attributed to the mobility of the organ. Against these views it is held that nephroptosis is not unconditionally associated with splanchnoptosis; that the great majority of cases of nephroptosis are independent of enteroptosis; that the patients who come under the observation of the surgeon on account of floating or movable kidney are, as a rule, free from undue mobility or ptosis of any other abdominal viscus; that the most severe renal and abdominal crises are cured by nephropexy; that women with lax abdominal walls, an evident degree of splanchnoptosis, and a freely movable kidney may be quite cured of all renal and intestinal symptoms by the operation; and that the classical gastro-intestinal and nervous symptoms are sometimes caused by a kidney moving in a "cinder-sifting" manner, the mobility of which cannot be detected by palpation. Experience has taught that there are cases of nephroptosis, others of enteroptosis, or splanchnoptosis, and others, again, in which nephroptosis and splanchnoptosis co-exist;

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that in many cases very severe symptoms are caused by the mobility and prolapse of the kidney; and that the treatment suitable for enteroptosis is not sufficient in many cases to relieve or even to influence the symptoms due to movable kidney. I am as sure of this as of any fact in surgery—namely, that the most complete and lasting relief from renal and abdominal crises has followed nephropexy; and that patients, after the operation, have improved in general health and spirits, have confessed themselves better than they had been for many years, have put on flesh, and have been pronounced by their relatives and friends as quite altered beings, who, however, had previously and in vain tried all kinds of treatment both at home and at health resorts.

The Diagnosis of Movable Kidney.

In no case are we justified in positively concluding that a patient's sufferings are due to movable kidney unless we can actually feel the kidney to be unduly movable. In cases in which one-sided pain, some amount of gastro-intestinal disorder, and occasional crises with possibly a little pus or blood in the urine, occur, though without any tangible signs of undue mobility of the kidney, we may suspect a movable kidney, but the diagnosis cannot be made absolute until the organ is explored. We must bear in mind that the "cinder-sifting" renal movements cause similar symptoms to those of nephroptosis generally, also that they cause symptoms closely resembling those of stone in the kidney or ureter as regards pain and renal crises, but that they will entirely escape detection if we lean too implicitly upon the results of the Röntgen rays, and if when we fail to find evidence of a stone, we decide not to explore the kidney. The same is true also of abnormalities of the renal pelvis and ureter, of early tuberculosis, and of small abscesses of the kidney, each of which, though remediable by operation, cannot be detected by the X-rays. On the other hand, even when all the typical symptoms are complained of, we must not decide that the movable kidney is the cause of them until we have made a careful investigation of the other organs of the abdomen and pelvis, both as to their anatomical character and relations, and as to the way in which their functions are discharged. By means of careful and systematic palpation the diagnosis between a "movable" or "floating" kidney on the one hand, and other movable abdominal

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tumours on the other hand, is usually sufficiently easy, although there are several instances of mistakes having been made by most capable clinicians. A "floating" kidney has been sometimes diagnosed as something else, but what happens far more frequently is that some other tumour, not renal, is diagnosed as a movable kidney. Probably the commonest error is to mistake an enlarged gall-bladder for a "floating" kidney. These two conditions often co-exist. A movable right lobe of the liver has been taken for a floating kidney, as have also a movable spleen, cancer of the cæcum, cancer of the stomach, a tongue-shaped lobe of the liver, an intra-peritoneal abscess, an ovarian cyst or dermoid of the ovary, a uterine myoma, and tumours of the omentum or mesentery.

Pathological Effects of Movable Kidney.

The most common effect of movable kidney is the general impairment of the health of the patient. Languor, debility, loss of flesh, vertigo, constipation, hypochondriasis, great restlessness, great anxiety, perversions of sensations or of the special senses, hysteria, and many other neurotic conditions are the ordinary consequences.

The most frequent effect upon the kidney is hydronephrosis, either intermittent or persistent. When distended a hydronephrotic organ may give rise to an abdominal tumour, but in other cases the kidney, though converted more or less into a mere sac, is of small size, and cannot be detected by palpation, even when tightly distended.

Treatment.

In many cases of movable kidney the symptoms, if any exist, are so slight that no treatment is requisite. When symptoms occur in spite of the avoidance of active exercises—when pain, aching, and a wearying sense of dragging weight or of great lassitude are caused by ordinary standing and walking, or when pain occurs even during absolute quietude and total avoidance of every form of physical exertion, nephropexy is called for.

Various forms of trusses and bandages have been devised, and have in some cases given some partial relief. But though some patients are rendered more or less comfortable, they are not really cured by these appliances. Moreover, the pads and bandages are not free from the risk of doing more harm than good in cases

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belonging to groups 1, 2, and 4, unless care is taken not to apply them before the kidney is replaced in its natural position, or if the kidney descends after the pad has been duly adjusted. Very much depends upon the care with which the belt or bandage is applied. I have known much comfort and an entire freedom from crises to be derived from their use during the time that the patient was under supervision and was not allowed to rise until the kidney had been replaced and the belt adjusted, but as soon as the patient was left to put on the bandage for herself all her troubles returned. One such patient suffered some of the most severe renal crises that I have ever witnessed, and had often to be kept under chloroform for three or four hours at a time. She was quite cured by nephropexy 12 years ago, and has led a very active life ever since. In fact, in cases of simple or uncomplicated nephroptosis belts and pads are practically useless, and some of the frightful things which have been produced under the name of "kidney pads" ought to be relegated to a chamber for the collection of instruments of torture. It is chiefly in the rarer cases in which movable kidney is associated with enteroptosis that belts are of any real use, and in these cases the best form of belt is one shaped so as to fit well above the hypogastrium and without any pad, to support the abdomen generally. Not a few women find a belt so irksome and uncomfortable that they positively refuse to continue wearing it, especially when they realise that they can never be cured by the treatment, that though the belt may keep the kidney in place by giving a general support to the abdomen, it cannot end in fixing the kidney in its proper place, and that to give continuing comfort it must be worn for life.

My experience leads me to attach next to no value to rest in the recumbent position. In several instances in which I have operated with most gratifying results the patients have kept the recumbent position for six or nine months before the operation, without the least improvement from it, all their symptoms returning immediately after getting about again.

An operation should certainly be advised when the symptoms are severe and are not relieved by rest, mechanical appliances, and appropriate medicinal and dietetic treatment; when mechanical appliances cannot be borne or seem to increase rather than to mitigate the symptoms; and when the patient cannot take gentle exercise or even sit erect for long without suffering. In cases in

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which, in spite of palliative treatment, paroxysms of nephritic colic, fainting, sickness, vomiting, and pain radiating far and wide in the course of the branches of the lumbar plexus of nerves occur frequently, the only possibility of relief is surgical operation.

The only operation justifiable is nephropexy. Nephrectomy is no longer admissible for movable kidney. Nephropexy has proved to be both safe and successful, and it has entirely superseded nephrectomy. Collected statistics show the average mortality of nephropexy to be but slightly over 1 per cent., and my own tables, giving all the operations of this nature which I had performed up to March, 1898, show 57 cases without a single death. Up to the present time, in considerably over 100 operations, I have had only one death, and that was from cardiac thrombosis in a stout female whose kidney was incised and explored before being fixed. Nephropexy has not the same value as a means of relieving each of the three leading classes of subjective symptoms—viz., (1) pain, (2) gastro-intestinal disturbances, and (3) nervous phenomena. It has cured 90 per cent. of the cases in which pain was the chief symptom. If pain continued after the operation it was because either the diagnosis was at fault or the kidney was otherwise deranged. In the gastro-intestinal group of symptoms, the operation has been less successful than when performed mainly on account of pain. Flatulent dyspepsia and constipation are the two symptoms of this group which I have most frequently seen to persist, but there have been very few patients amongst those I have operated upon, and whose subsequent histories I have been enabled to follow, who have not been improved even in these respects. On the nervous phenomena the effect of the operation is far less satisfactory. Operations upon hysterical patients for the relief of subjective symptoms, even when they are based upon a real ascertained physical basis, are not only apt to fail in giving relief, but may arouse ideas which end in fresh complaints, or in an aggravation of the original ones. If, however, the hysteria or neurasthenia has been secondary to the movable kidney we should not hesitate, because of the uncertainty of the therapeutic effect to operate, even though the nervous symptoms are very pronounced. If palliative means have been found insufficient we should perform nephropexy, though we should warn the friends, if not the patient herself, of the speculative nature of the result.

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There are several ways of performing nephropexy, and many surgeons prefer some one to all the others. Practice has taught me that it is best not to confine oneself to one form of operation. There are at least three which have special advantages in different kinds of cases. I choose one method in one case, and another method in another. The methods which I employ are: first, Vulliet's operation; secondly, that which I call my own; and thirdly, a modification of the method recommended by Tuffier.

The conclusions as to the treatment of movable and floating kidney by nephropexy at which I have arrived are the following. I would, however, preface them by repeating what I have said above respecting belts and bandages—namely, that they have no efficacy in nephroptosis uncomplicated with enteroptosis, and that, when employed in cases of movable kidney, they increase pain, and may otherwise do harm if not carefully and properly applied:—

1. When movable kidney is associated with enteroptosis, no operation should be performed on the kidney unless it is evident that the more serious symptoms are due to the mobile kidney alone, and not until after the trial of a well-fitting abdominal support, and the careful dietetic and medicinal treatment of the gastric and intestinal disorders. Should these means fail, and the kidney evidently be most at fault, nephropexy, followed by the wearing of an abdominal belt, should be tried.

2. When a movable kidney is complicated by a movable liver, or when both kidneys move, the same rule should be followed as in general enteroptosis; in the case of both kidneys moving (when both organs have been giving trouble), they should be fixed one after the other at an interval of a week, so that convalescence from both operations may be taking place simultaneously. I have, in several instances, thus operated upon both organs with the most satisfactory results.

3. When the movable kidney occurs in a hysterical or neurasthenic patient, all palliative means should be tried before resorting to an operation, and the patient's friends should be informed of the uncertainty of the result from operation. The statistics show that a cure may be hoped for by nephropexy in about half these cases.

4. For uncomplicated movable or floating kidney, in which the principal symptoms are pain and gastro-intestinal troubles, the operation may be confidently advised and carried out without any previous trial of belts or of rest.

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5. When renal crises are a feature of the case nephropexy ought to be strongly urged, because of the impossibility of keeping the kidney in its proper place by a belt, and because of the constant risk of hydronephrosis and recurring pain, even if the renal crisis can be kept under control.

6. When a movable kidney gives rise to no inconvenience an operation ought not to be thought of, and a belt need not be worn.

The CHAIRMAN (Mr. John H. Morgan) said that, owing to the unavoidable absence of the President, he found himself in the responsible position of chairman that evening without having had any warning, and, therefore, he was not prepared for taking part in a discussion of the subject which had been laid before the meeting by so eminent an authority as Mr. Henry Morris. A glance at the synopsis of the paper would show that there was a great deal to be discussed. It was a subject in which both surgeons and physicians were deeply interested, because it frequently happened that instances of the condition came into the hands of the physician before they were transferred to the wards or the hands of the surgeon. Much that Mr. Morris had said had thrown light on phases of the subject with which he, Mr. Morgan, for one, was far from familiar. One of the great difficulties he had always found in dealing with these cases was that of ascertaining how far reliance could be placed on the patient's own statements. There were a number of people who came for advice with all the symptoms of floating or movable kidney, who were of a very neurotic type, and to distinguish in them between what was true and what untrue was very difficult. Mr. Morris had done a great service in pointing out the cases in which operation was really likely to be of value. Undoubtedly many of the so-called failures were due to the fact that those who operated had not given full consideration to the various aspects of the case, and had been hasty in operating. He meant that surgeons sometimes ascribed to mobility of the kidney symptoms which, as Mr. Morris had pointed out, were more likely to be due to enteroptosis, which so often co-existed. He agreed with Mr. Morris that rest was of little or no value in the cases under consideration. He also had found that operation generally relieved all the symptoms. He had also met with one or two cases in which, after operation, the condition had recurred, but possibly this was due to the fact that the procedure in these cases had not been quite so up-to-date as that practised by Mr. Morris. He could have wished that Mr. Morris had had time to detail the procedure which he followed in the three forms of operation which he had described. Perhaps he would rectify that deficiency in his reply.

He could not conceive anything more admirable than the conclusions with which Mr. Morris terminated his paper. They summed up the present knowledge of the subject, and embodied Mr. Morris's own great experience.

Dr. MACLAGAN said that perhaps from a medical point of view he might be allowed to make some remarks. He supported the tribute of the Chairman as to the merits of Mr. Morris's paper, and the complete and thorough manner in which he had dealt with the subject. He differed from Mr. Morris on two points, and on one from the Chairman. Both

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the reader and the Chairman said they had never found any good come from rest. One of his patients had suffered from all the symptoms of movable kidney—constant dyspepsia, irritation, and a little yellowness, not actual jaundice, and disturbance of the liver and stomach. She had been to Karlsbad, to Harrogate, and other places, and had had all sorts of treatment before coming under his notice. She was thin and worn by the frequent pain, indigestion, and malnutrition. He contemplated the possibility of operation, and put her to bed. But he kept her there for six weeks and had her regularly massaged, at the same time feeding her well. She improved enormously in that time. That happened one and half years ago, since which she had never had any symptoms, and he had not been able to feel her kidney move since. Another patient was sent to him from Uxbridge, whose kidney moved, and there was disturbance of the liver and a general sense of misery. He recommended her doctor to put her to bed, carry out massage, and feed her well. He did so, and she got perfectly well, and has remained so ever since. Many of the cases of movable kidney occurred in women who were thin and emaciated; under rest and feeding the condition improved, and fat was deposited over the body, the deposit around the kidney sufficing to keep that organ in place. To attain this end the patient should be kept on his back. Again, Mr. Morris said that jaundice was never due to pressure on the bile ducts in movable kidney. Two years ago, he, Dr. Maclagan, recorded in the 'Lancet' three cases of jaundice, hepatic colic, and all the symptoms associated with gallstones, due to pressure on the cystic duct of a movable kidney. One of those cases had undergone the usual treatment for hepatic colic. She was sent back from Karlsbad with the information that she had a gallstone in her bladder and that she would not be well until it was removed. Mr. Knowsley Thornton saw the patient with him, and the kidney was felt to be distinctly movable. There was tenderness over the gallbladder, and an operation was thought advisable. She had the usual operation in front for the removal of gallstones, but nothing was found in the gallbladder but bile. The kidney was felt behind it, and was fixed to the loin. The second case was one of typical hepatic colic, and he saw the lady when she was coming out of one of the attacks. She was jaundiced, had a high temperature, and a great deal of pain, which had been relieved by morphine. She had passed a gallstone at one time. A year and a half afterwards he saw her in London in a similar attack. She was advised by Mr. Treves and himself that something must be done, as latterly the attacks had occurred fortnightly. At the operation no gallstones were found, but a tumour was felt behind the gallbladder. That was found to be the kidney, and Mr. Treves replaced the gallbladder and fixed the kidney. The third patient had never passed a gallstone, but otherwise the case was similar to that previously mentioned. In her case eventually also he insisted upon an operation. When that was carried out, exactly the same thing was found. All those cases were now perfectly well, and tended rather to controvert what Mr. Morris had laid down in that respect.

With regard to neurasthenia, if there were a movable kidney in a woman with an extremely susceptible nervous system, more definite symptoms would be complained of in such a patient.

Mr. Bruce Clarke said that there were one or two things which should be borne in mind when one heard the physician state his views and the surgeon his—namely, that the physician saw the earlier stages of the cases, whereas the surgeon was not called in until the physician,

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under whose care the patient was, had made up his mind that an operation ought to be performed. He had often seen cases in which he was told of symptoms which had occurred, but which he personally never actually witnessed in the course of the case so long as it was under his care. It was interesting to hear how some cases of movable kidney had benefited by rest, but he believed such cases were those that were associated with considerable enteroptosis; in other words, they were not *bonâ fide* cases of simple movable kidney, but movable kidney complicated by enteroptosis. One of the most important things was to come to a conclusion as to what one meant by movable kidney. He knew nothing on which people differed so much as on that point. What one would call a movable kidney, another might say was not worthy the name. In an ordinary individual the kidney moved up and down with respiration as much as the liver did. One symptom only, he thought, would enable them to decide whether the condition of the movable kidney was of any gravity—namely, when it caused pain. Many such kidneys passed through a stage of painfulness, and then afterwards ceased to cause pain, simply because they could move about so freely. What was the cause of the acute crises of pain in connection with movable kidney? He thought it chiefly due to the retention of urine in the pelvis of the kidney, just as there was pain from retention of urine in the bladder. Mr. Morris had referred to the paper which he, Mr. Bruce Clarke, brought before the Royal Medical and Chirurgical Society on the subject, when he described cases of dislocation. He had seen many cases since, in which those symptoms could be relieved by pressing upon the kidney and so getting it to void its contents. Whether dislocation was the best term to use for such a condition was comparatively a small matter.

Another point was the association of those kidney cases with digestive troubles. Why were they so associated? He thought that by some normal or abnormal connection taking place between the duodenum and the kidney, whenever the kidney moved downwards and out of its place it pulled upon the stomach, and so caused those attacks of vomiting which one saw from time to time. One such patient he had seen was literally reduced to a shadow. In one of her attacks she fell out of bed on a cold night, and was found the next morning half dead with cold under the bed. He stitched her kidney, and she was now perfectly able to walk three or four miles, and enjoyed a fairly reasonable amount of health, though he could not describe her as robust.

As to what sort of operation should be performed, he hoped Mr. Morris would describe the exact method. He thought it most important that the kidney should be got out of its bed of fat, and care should be taken that there was no fat between the kidney and the lumbar fascia behind. The particular method of stitching the kidney did not matter, so long as that precaution was taken. In one or two cases he had had to remove the kidney that had been movable, because he had tried, by nephropexis, to save an organ which was in a state of suppuration. The difficulty of getting it out had been very considerable, and one had almost to make a sort of artificial separation between the kidney and the lumbar fascia. He was sure that the more experience one had of those cases the more absolutely certain one would be that they were real cases, and not instances of hysteria or neurasthenia, as some people said. He strongly objected to the term neurosis. He believed the best definition of neurosis was a tendency in the individual to unduly exaggerate ordinary symptoms.

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Mr. SWINFORD EDWARDS said he also would like to hear from Mr. Morris what operation for performing nephropexy he favoured. Whatever was done, the most important thing was to replace the kidney. He had seen cases where the kidney was only partially replaced; true, it was sewn to the integuments in the lumbar region, but the upper part of the kidney remained on a lower level than normal. It was easy to stitch a kidney between the crest of the ileum and the last rib, but not so easy to put in the upper sutures on a level with, or higher than, the last rib. He would also like to hear whether Mr. Morris had encountered cases of hydronephrosis following nephropexy. He came across such a case the other day, in which a surgeon had performed nephropexy two months previously for the relief of a movable kidney, and in which hydronephrosis followed. To such an extent did that take place that he, Mr. Edwards, had to do a nephrectomy, for he found that the pressure, due to the retained urine, had been so great that very little renal substance remained. The only case which had relapsed, in his series of nephropexies, was that of a young woman in whom the nephropexy held good for six months. She was completely relieved of all her symptoms at the time, but after that period she returned with a lump in her belly which was evidently the kidney. It was very painful, and on that occasion he could not reduce it. After remaining in bed for a week, it went back and could not be felt, nor could he dislocate it. But the same thing happened again, and it could not be reduced, though after a period the tumour again vanished. He could not quite explain those phenomena. The urine she was passing at that time did not differ from the ordinary, and the kidney did not feel as if it was hydronephrotic. She promised to come up again for a further operation, but as yet had not done so.

Mr. BIDWELL said he would be glad to hear the ultimate results of the operations. It had been several times remarked to him that surgeons recommended nephropexy, but did not know what happened to the patients some years afterwards. He had taken considerable trouble to trace his own cases, and had found that after an interval of two or three years several of the patients seemed to have complained of pain again, and that without any further dislocation of the kidney. The method he had employed in his last 20 cases had been that of Vulliet, since this ensured that the kidney was fixed in a proper position. He agreed it was especially in the cases where neurasthenia was present that recurrence of the trouble occurred.

Mr. WALTER SPENCER said he thought there were two sets of cases, which were difficult to distinguish—those which, after having had a distinctly movable kidney for some time, had some slight hydronephrosis, and in other cases a collection of urates or gravel. He had had to operate on two or three cases in which the kidney had been previously firmly fixed in the loin, but with a continuance of painful symptoms. In fact, some cases of movable kidney required the further operation of nephrotomy in order to relieve them. In those cases he had thought it right while fixing the kidney, to do, at the same time, nephrotomy into the pelvis of the kidney, and drain for some time by keeping in a strip of gauze. There was no further complication, the wound healed in about a month quite satisfactorily. These cases of movable kidney, with hydronephrosis or collections of urates, showed very slight symptoms, and so were difficult to detect. But he felt sure that if nephrotomy and temporary drainage were more often combined with nephropexy there would be better results.

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Mr. Morris, in reply, said that with regard to the curative effect of rest it was a matter of individual experience, but that the cases mentioned were probably cases of enteroptosis. He believed that a deficiency of the perinephritic fat tended to the production of movable kidney, and Dr. Maclagan's cases seemed to support this view. With regard to the cases of jaundice produced by movable kidney, he said that he did not think that they were due to the direct pressure on the biliary passages, but they might be due to dragging on the duodenum, and thus producing obstruction of the duct at its entrance into the intestinal canal. He thought that the increase in the size of the kidney was due, not to hydronephrosis, but to venous congestion due to obstruction of the veins. He mentioned a case in which he had at first fixed a hydronephrotic kidney, but four months later he had to remove this owing to a recurrence of pain. He had never seen hydronephrosis follow an operation. With regard to after-results, he hoped he had made it clear in his paper that the cases in which symptoms returned were in the neurasthenic patients. He described Vulliet's operation, which was, he said, unnecessarily severe, and he did not think it advisable either in fat or in neurotic patients, but it had the advantage of drawing the kidney up under the ribs.

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December 9th, 1901.

VOTE OF CONDOLENCE TO LADY MACCORMAC.

THE PRESIDENT: For a few moments, gentlemen, I must ask your permission to interrupt the ordinary process of the business of the meeting in the discharge of a very sad duty. A few hours ago we laid all that was mortal of Sir William MacCormac in his grave. For 30 years exactly Sir William was a Fellow of this Society. Within that time he performed the duties of Councillor, was an Orator, a Vice-President, and, 13 years ago, he occupied the Presidential Chair. Such a record in itself might almost be deemed worthy of a claim to recognition by a few words on an occasion such as this, the more so because I believe this is the only Society of all the medical societies in London in which Sir William held any active position. It is not for me to attempt to appraise the work which Sir William MacCormac did, or the position which he took in that particular branch of the profession which he followed. There are others here who could do that far more fittingly than I can. And I scarcely think that it would be possible for anyone, whoever he might be, to claim definitely to affirm the status of any one of his working brethren; time and the judgment of his peers alone will do that. It was probably in the pre-eminently public position which Sir William MacCormac took in reference to his profession that he will be, as he is, best known. Giving up an assured position in his native town, he entered on that wider sphere of labour in London, to which he came with all the prestige which attached to the excellent work that he did in the battlefields of Europe in connection with the Red Cross. His association with St. Thomas's, one of the large Medical Schools of London, as a surgeon and teacher, as well as an examiner in the University of London and in the College of Surgeons, afforded the opportunity of his fulfilling very much the same kind of duties that others in his position have to do. But in addition to all that, he took a prominent public position, and we know the way in which he discharged the duties of Chief Secretary to the Congress in London in 1881. There is also the fact that he held, for five years—the longest tenure of office, I believe, in the century—the Presidency

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of the College of Surgeons, during which time he conducted with remarkable success the celebration of the Centenary of that Institution. His connection with the Court and his recent labours in South Africa in the early time of the war are known to you all. He was keenly alive to the appreciation of his work, whether by public approval or by the bestowal of the honours which crowded in on him. And thus he led a busy life. But it is not for these reasons only that the memory of Sir William MacCormac will remain. Those, and they were many, who were privileged to enjoy the intimacy of his friendship would feel that any reference to his name were incomplete and inadequate, did it not allude to the lovable personality of himself. Privileged as I was to enjoy that friendship during the past few years very closely, and still more connected within the past few weeks, in association with Dr. Anderson, in endeavouring—all too unsuccessfully—to minister some relief from the pain and suffering which he experienced, I may be forgiven if I feel that I have sustained in his death a personal loss. Long will be remembered his commanding presence, his genial personality, his open-handed generosity; but for the future they will be to us merely recollections.

Acting upon a suggestion that was made to me, and with such time as was allowed, in consultation with Fellows of the Society whom I had the opportunity of meeting, I, with the sanction of the Treasurer, sent to his funeral, in the name of the Society, a wreath. I trust that that will meet with your approval, and I beg to propose this resolution:—

> "That the Medical Society of London, having heard with deep regret of the death of their former President, Sir William MacCormac, Baronet, desire to place on record their high appreciation of his public services to the profession and to the Society, and, further, to convey to Lady MacCormac the respectful expression of their sympathy."

The resolution was carried in silence.

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THE MEASURE OF PHYSICAL FITNESS FOR LIFE IN THE TROPICS.

By ALEXANDER CROMBIE, M.D. Edin., Lieut.-Colonel I.M.S. (Retd.).

MR. PRESIDENT AND GENTLEMEN,—Dr. Andrew Duncan, in his excellent work on 'Campaigns in Hot Climates,' points out very clearly that in such undertakings, climate, as climate, takes a very small part in causing the sickness of armies operating within the tropics, and quotes Dickson, late of the Royal Navy, to show that "a high average of health and efficiency can be maintained *for a time*, in every kind of geographical position." It is not the climatic conditions, to which man appears to be able to accommodate his physiological processes with remarkable ease, which cause the sickness and mortality, but the accidents, the evil chances, the diseases incidental, but not essential, to life in the tropics, which cause the disastrous results so frequently noted. If the parasites of cholera, dysentery, enteric fever, and malaria could be eliminated, the health of troops campaigning within the tropics would probably compare favourably with that of similar bodies of men engaged in like operations in temperate latitudes *for a time*. Ephemeral, and simple continued fever (properly so-called), and heat apoplexy, would no doubt take the place of the attacks of bronchitis and pneumonia of the temperate zones, but without any very marked increase of general sickness or mortality; but, mark the qualification, in the course of time there would probably arise in Europeans living in such altered climatic conditions a perceptible deterioration of health, especially in those least able to adapt themselves to the new conditions, by reason of defects of constitution or health, which rendered them less adaptable. It is with these defects of constitution and health, and not with the diseases of the tropics, that I propose to concern myself to-night, and as it will be seen, in the course of my remarks, that the heat and moisture of such climates seem to suit some constitutions better than the rigour of colder countries, I have ventured to alter the title of my paper from that of "The Physical Disabilities for Tropical Life," to its more general converse, namely, "The Measure of Physical Fitness for Life in the Tropics."

In order to give clearness, method, and some kind of logical sequence to my remarks, I will ask you to bear with me while

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I pass in review some of the modifications which the altered conditions of warmth and moisture cause in the physiological processes normal to an organism trained in a temperate climate, when more or less suddenly transferred to a tropical one, because it is on these modifications, and the varying power of accommodation to them in different individuals, that the fitness or unfitness of one man more than another to keep his health in the tropics depends.

Alteration in Thermogenesis.

It is obvious that transfer from a temperate to a hot climate must, in the first place, alter very materially the balance between thermogenesis and thermolysis, by means of which the body temperature is maintained within physiological limits. In temperate regions the heat-producing processes are required to raise the temperature of the body 30°, 40°, or 50° F. above that of the medium in which it is placed; in a tropical climate they may not be required to do so at all, or only 5°, 10°, or 15° F., according to region and season. As the process of thermogenesis is largely one of combustion, it follows that in hot climates there is a marked reduction in the waste products of combustion, of hydro-carbons exhaled from the lungs, and of the nitrogenous elements of tissue and food eliminated by the kidneys.

The amount of carbonic acid given off by the lungs in the tropics is reduced by about 20 per cent. as compared with the European standard, and the quantity of carbon burned to produce the amount of carbonic acid given off is lowered from 10 to about 8 ounces per diem. Similarly there is a marked reduction in the quantity of watery vapour—the other product of hydro-carbon combustion—exhaled by the lungs to the extent of about 4½ per cent., the diminution being represented by about 6½ ounces of water daily (Rattray).

This reduction in the activity of the respiratory function required in hot climates brings with it other consequent changes. These are :—A smaller number of respirations per minute ; reduced vascularity of the lungs amounting to about 12 to 13 fluid ounces of blood. Therefore the lungs are together lighter by about three-quarters of a pound. And the capacity of the lungs for air is in Europeans increased to a corresponding degree, *i.e.*, about 30 cubic inches. This lowered demand on the respiratory function has

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resulted, in natives of hot climates at least, in the equable, hot, moist parts of India, in the evolution of a race of comparatively narrow-chested men.

Pflüger, on the other hand, found that when the temperature of the air reached 95° F. or more, the pulmonary exchange was increased instead of being diminished, and it is necessary to bear this in mind when we come to speak of the effect of sustained high temperatures on the human economy.

It follows from this lessened call on the activity of the lungs, that inflammatory diseases of the chest are less frequent and less severe in tropical than in temperate climates. The more equable climatic conditions, as well as the comparative infrequency of the respiratory movements, the increased vital capacity and diminished vascularity of the lungs, all contribute to this result. It follows from these favourable conditions also that people with weak lungs, and therefore subject to congestion and inflammation of the pulmonary mucous membrane, do well in an equable tropical climate, such as is found in islands and within reach of the sea, but it is otherwise in the interior of tropical continents and where there are extremes of temperature. There the daily fluctuation of atmospheric temperature is apt to be great, and there is often much dust, and inflammatory diseases of the chest are frequent and severe. Thus, speaking generally, pneumonia and respiratory diseases altogether are less prevalent in the equable climates of Burma, Bengal, and Southern India, than in the inland stations, and especially in the hills, and in the Indus Valley, where in winter the extremes of temperature are greatest, these diseases reach a maximum prevalence and severity. This is especially so with natives and others of debilitated constitutions and lessened power of resistance to infection.

I will reserve what I have to say of pulmonary phthisis to a later part of this paper.

As the lungs are to the products of combustion of hydro-carbons, so are the kidneys to the nitrogenous elements of the tissues and food. As the diminished metabolism demanded by the warmth of the atmosphere lessens the excretory work of the lungs, so does it that of the kidneys. The quantity of urine excreted in 24 hours is rather less, about 1,250 cc. in such a climate as that of Lower Bengal, where I make my observations, against 1,500 in temperate climates, probably because the larger quantities of fluid imbibed

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nearly counterbalance the excessive perspiration. The specific gravity is, however, much lower. It seldom in Europeans exceeds 1,016, and 1,015 to 1,018 may be taken as the average; and in natives, with their simpler and less nitrogenous diet, it is generally much lower, and may be said to range in health from 1,008 to 1,014. Just as with the lungs, which are called into greater activity by very hot weather, so one frequently finds a large quantity of urine of very low specific gravity passed at the hot season, contrary to what one would have expected. Especially is this so in the delicate organisations of young children; and it was not rare to have an epidemic of hydruria among European children in the months of April and May in Calcutta; and in succeeding members of the same family in successive years. This condition was usually ushered in by a low febrile state, and I regarded the whole phenomena as an expression of nervous exhaustion consequent on the heat.

In very hot weather, however, especially when the atmospheric conditions are favourable to evaporation from the skin, the urine is apt to become scanty, and of high specific gravity from concentration, often reaching close on 1,030, whether, however, with any increase in the amount of urea excreted I do not know. These are just the conditions which prevail in those districts of India where stone in the bladder is so frequent, and this concentration of the urine in the hot weather is probably the important factor in starting the formation of these concretions.

The more usual low specific gravity of the urine in the tropics may be taken as a measure of the diminished formation and excretion of urea, uric acid, &c., derived from waste of nitrogenous tissues and food, and taking the figures I have already mentioned, this would amount in Europeans to about 22·3 grammes, and in natives to about 17·2 grammes of urea in 24 hours, as against 33 in European countries, *i.e.*, it is reduced by about a third in Europeans and nearly a half in natives. It follows from these considerations that people subject to disorders of the secretion of the kidneys, or to the abnormal formation of uric acid in the system, do well in the tropics so long as they live in an equable climate. Gout in its typical acute manifestations is comparatively rare in Europeans in the tropics, even when they are of a gouty family, and have had one or more attacks before leaving England; and it is very seldom seen in god-fearing natives who adhere to the rules of their religion in

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the matters of diet and alcohol. In the European, who has become exhausted by long residence in a warm climate, and has indulged freely in the good things of this life, which, unfortunately, are not made of vegetables, and especially if he has inherited a predisposition to gout, the irregular forms of that diathesis are by no means rare. It then takes the form of gouty eczema, especially of the legs and scalp, of acid and flatulent dyspepsia, and congestion of the liver; and in younger men with a strong hereditary taint, of headaches, with high blood tension, and inability to stand exposure to the sun.

It is also a corollary of the comparatively lower call on the activity of the kidneys that albuminurics do well in warm, equable climates. The disease follows a milder and slower course, and reaches the secondary changes and the uræmic stage later, but they do not get cured any more readily. Acute Bright's disease is, on the other hand, more amenable to treatment than in cold climates. Chronic Bright's disease is very common in natives past middle life.

Acute rheumatism, which it is convenient to consider here, whatever may be its true pathology, is also rare in the tropics, and I am not aware that I ever treated a case of this disease in a European during my 25 years' residence in India. According to the Army returns, rheumatic fever is most frequent in the dry, central parts of India, and the very wet west coast. It is not mentioned in those of the native army, or jails. It is above all most frequent in the hills. I used, however, to see acute, or rather subacute, attacks of rheumatic fever with considerable frequency among the well-to-do classes of natives in Calcutta, with all the usual cardiac and other complications.

Muscular rheumatism, whether it has any relation to the true rheumatic diathesis or not, is probably just as common in the tropics as at home.

No one should send a case of osteo-arthritis to a hot, moist climate, in the hope that it will improve in the warmer conditions. These cases, according to my experience, do badly in such a climate and improve on returning to Europe. Ordinary chronic articular rheumatism is, on the other hand, improved by residence in the tropics.

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THERMOLYSIS.

Turning now to the processes by which the temperature of the body is prevented in the tropics from rising above a point compatible with the healthy performance of its functions, we may dismiss the minor losses of heat which occur in the excretion of urine and fæces, and in the respiratory function, and confine our attention to that through the skin, by means of which about 80 per cent. of the work is done. By evaporation from the skin about 15 per cent. of the loss of heat from the body takes place in temperate climates, and about 70 per cent. by radiation and conduction from the surface. As the temperature of the atmosphere rises the loss by radiation and conduction becomes less and less, until when the atmospheric temperature approaches and exceeds that of the body it ceases altogether; while on the other hand with every rise of atmospheric temperature the sweat glands become more active, and at last evaporation from the surface is practically the only remaining physical means by which the normal temperature of the body is maintained. The skin pours forth fluid which in dry, hot climates evaporates as fast as it is formed, and in warm, moist ones, like that of Lower Bengal, forms beads of perspiration which trickle down to lower levels, and it is in the latter circumstances that evaporation is so much facilitated by keeping the air in motion by means of punkahs. So great is the cooling effect of the evaporation of this excessive perspiration, that the skin in the tropics often feels cold and clammy, and this is so, not only in health, but even in active febrile conditions, where the skin may feel quite cool to the touch while the temperature of the mouth may be 104° F.—a most deceptive state of matters. The effect on the body temperature may be estimated when you consider that to supply the material from which so great a quantity of fluid may be derived, a large quantity of blood must be directed to the surface of the body, which in temperate climates is directed elsewhere, chiefly to the internal organs; while in the tropics in a few minutes many pints of blood are subjected to the influence of this cooling process at the surface, and then collected in the veins and distributed by the heart to cool the rest of the body.

It follows from this constant hyperæmia and moisture of the skin that this forms a most favourable surface for the cultivation of vegetable parasites, and all parasitic and exudative diseases of the

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skin are very prevalent in the tropics and difficult of cure, especially when unsuitable, warm, absorbent, non-conducting clothing is worn which interferes with free evaporation. Of these diseases prickly heat and *tinea circinata* are the most common, and very few Europeans living in the humid climate of Lower Bengal are ever long free from them. Eczema and *impetigo folliculorum* are also frequent, and crops of distressing boils, due to the presence of the ubiquitous streptococcus (I have opened 25 on the forehead at a single sitting), are a constant cause of illness and invaliding. Carbuncles of enormous dimensions (we measure them by soup plates), especially in diabetics, are quite a feature of tropical practice. Dry, scaly eruptions, on the other hand, are rare, and I do not remember having seen more than one case of psoriasis in the whole of my tropical experience, and I think it would be quite worth while for practitioners in this country to try the curative effect of a short residence in a warm, moist tropical climate in the treatment of obstinate cases of this kind.

Another result of this habitual hyperæmia of the skin is the great sensitiveness of the European, who is living or has lived in the tropics, to chills. A fall of temperature from 90° to 80° F., will produce a feeling of chilliness out of all proportion to the actual temperature, with consequent contraction of the cutaneous capillaries, which sends the blood in on the internal organs with congestion of the liver and spleen, and if he has any malarial parasites lurking in his spleen or the marrow of his bones it wakes them up into activity. As a consequence of this sensitiveness of the cutaneous surface no Anglo-Indian can stand a cold bath without serious results to the liver and digestive organs generally, and many cases of this nature are cured at once by resorting to warm instead of cold water. It follows from this that no European of delicate constitution easily upset by changes of temperature should go to the tropics even if free from all organic disease. The hardiest men are probably those who have been born on the east coast of Scotland, and have become inured to, and survived, its east winds and sudden changes of temperature.

On the other hand, many people of feeble circulation, whose powers of thermogenesis are weak, and who have a difficulty in maintaining their normal warmth in cold climates, do well in the tropics. Their feeble ability to produce heat is relieved of that tax in a warm climate, and they become more energetic in every respect

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for a time, before they feel the exhausting effects which sooner or later supervene.

The Inhibition of Thermogenesis.

But you will readily admit that there are atmospheric conditions in which these cooling processes at the surface of the body become difficult or impossible of accomplishment. When the temperature of the air rises to or above that of the body, and it is at the same time saturated with moisture, neither radiation nor evaporation from the surface of the body can take place; and there are many tropical regions where such conditions prevail for weeks or months every year. The feeling of distress which results is very great, and no one who experienced and survived the third week of June, 1888, in Calcutta, is ever likely to forget it. All business was suspended, the courts were closed, the tickagharries (hired cabs) refused to ply, and the tram-horses died at the rate of 18 or 20 a day. Even the long-suffering bullock fell down and died under the yoke.

How is the mean body temperature maintained within normal limits under such conditions? A corpse would have a temperature of about 100° F., and in the living body physiological processes are going on which are capable alone of raising the temperature of the body 10° or 20° F., and all radiation from the body necessarily ceases at temperatures of 100° F. and over, and in air saturated with moisture the cooling effect of evaporation from the surface of the body is reduced to a minimum, and probably does not exceed two or three degrees. What is the agency which prevents, under such conditions, the body temperature from rising 20 or 30 degrees above physiological limits? I cannot answer that question with any degree of certitude; but that there is some inhibitory influence at work must be admitted, and, moreover, we can conceive no influence working in this manner except a nervous one. As is well known, operations on the central parts of the nervous system cause deviations from normal temperature in the parts supplied by nerves from those centres; and when in some diseases, as cholera and yellow fever, all nervous control over the production of heat is removed by death, the temperature of the body undergoes a rapid rise. I have myself registered a temperature of 109° F. two hours after death from cholera, when the temperature of the air was not more than 80° F.

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Even in health so great is the difficulty which this great central nervous control has in keeping the temperature of the body in the tropics within physiological limits, that it is never quite normal if the temperature in cold climates be taken as the standard of health. The mean temperature of a European living in the tropics is always about half a degree higher than it is in Europe, and the least cause which tends to increase thermogenesis, when the heat and moisture bring physical thermolysis to a standstill, such as the use of the muscles in exercise, sends up the temperature to the very border of the physiological limit. I have generally found my own temperature well over 99° F. after exercise, and frequently close to 100° F., under such meteorological conditions as I am describing. Very frequently, indeed, the physiological limit is overstepped, and in marches, when men are wearing close-fitting uniforms and carrying weights, the central control often fails, and they fall out with febrile symptoms usually of a slight and ephemeral character; and this, and not ague, is by far the commonest form of fever met with in India. But sometimes under such conditions the central nervous control gives way so completely that the temperature runs up to 107°, 108°, and 109° F., and the condition is then what is called "heat apoplexy." Their companions in undress and not carrying rifles and knapsacks marching alongside, though flushed and hot, remain within the limits of physiological temperatures, showing how difficult it is and how great a strain there is on the nervous control to annul the effects of the heat-producing processes, which are necessarily increased by exertion, when so slight a difference in the conditions as the exertion of carrying a rifle makes all the difference in the ability of the nervous control to restrain the normal effects of unavoidable thermogenesis in conditions which are unfavourable to the loss of heat from the surface.

And this brings me to what I have been aiming at. In the tropics there is going on continually and unconsciously a tax on the nervous system which is absent in temperate climates. The nervous system, especially those parts of it which regulate the temperature of the body, are always on the strain, and the result is that in time it suffers more or less exhaustion. Every one who has lived in the tropics becomes conscious of this, and the longer he lives there the more conscious does he become of it. At first the sensation of heat is rather exhilarating, but before long the nervous

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system becomes exhausted by the calls made upon it. Perhaps the earliest and commonest signs of this exhaustion are a tendency to drowsiness, to drop off to sleep in the middle of dinner, or of reading a letter, or to go to sleep instantly on lying down for a few minutes in the middle of the day's work. Another is loss of all desire to eat, while there is at the same time a feeling of emptiness and want of food, and then a stimulant, a glass of champagne at the beginning of dinner, makes a new man of you in two minutes, and you fall to and eat with pleasure and profit. Here comes, to the European, the yearning for alcohol, and in the native for the, to him, far less harmful stimulation of opium. By-and-bye follows an inability to fix your mind on the subject in hand, failure to do simple sums of arithmetic correctly, loss of "nerve," forgetfulness of things that happened yesterday, irritability of temper, insomnia, and all the other phenomena of neurasthenia. This sequence of events is, of course, more likely to come on when prolonged high temperatures are combined with over-work and great responsibility, or when other conditions are unfavourable to the stability of the nervous system, such as depression from loneliness, anxiety, the destruction of many red blood corpuscles by the malarial parasite, and other debilitating illnesses.

Now there are especially two sets of conditions which make this work of controlling the body temperature more than usually difficult, and two sets of men who are more likely than others to fail in their response to the calls made on their nervous system. The first of these adverse conditions is obesity. Here there is originally no fault of the nervous system, but the mass of the body being disproportioned to the extent of surface by which the physical process of cooling takes place, the demand on the controlling centre is greater than in men of more usual proportions. The way in which stout men suffer from the heat is distressing to witness, and they are, moreover, most liable under any kind of stress to nerve failure, and especially to failure of the over-loaded heart. One therefore always attends a case of high temperature in a stout person with considerable anxiety; and we load them when they come for life assurance. The obese should not, for these reasons, go to the tropics.

The second class of cases under this heading which should avoid residence in the tropics is composed of those whose nervous system is naturally, or from any cause, weak, debilitated, or unstable.

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With such persons the nervous system succumbs sooner or later to the conditions under which it is called upon to work in hot climates—conditions not only of climate, but of occupation. Under the continued strain which I have endeavoured to explain, the central nervous system of the neurotic gives way; and there are two departments of Government service in India which especially illustrate these remarks. I refer to the Civil Service and the Forest Department. With regard to the former of these, I have no statistics to place before you, but the frequency with which men in this department break down mentally is a matter of common experience. They are often men of fine physique and muscular development, thanks to the public school and university life under which they have mostly grown up; but the strain on their nervous power produced by the severe mental training which they are obliged to undergo in order to gain a place in the list of successful candidates very often leaves its results permanently evident. They are peculiarly liable to loss of mental balance, especially in the early years of their service in India, and this is particularly marked in those who inherit the weaknesses of a neurotic family. The results of the severe and prolonged mental strain imposed on them before entering the service, followed by being early placed in positions of power and responsibility, together with long hours of work in an enervating, hot climate, produce unstable mental equilibrium in a certain proportion, and in not a few complete mental breakdown after a few years' residence in India.

I do not know whether the medical gentlemen who examine for the Civil Service Commissioners are aware of the frequency of such results among those they pass for the Indian Civil Service, but we of the Medical Board at the India Office are very much on our guard with reference to another department, which shows a similar frequency of mental breakdown. I refer to the Forest Department. The statistics of this department show that nearly 10 per per cent. die before attaining 10 years' service. Of 21 deaths, four, or 19 per cent., were due to mental symptoms; and of nine retirements, three were necessitated by insanity. The causes for this frequency of mental breakdown in these officers are not far to seek. They are the malarial surroundings in which they spend their time, with consequent destruction of blood corpuscles in large numbers, and frequent attacks of fever; their great exposure to the heat and sun during the hot season, the season of forest fires; and above all,

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the solitariness of their lives. It is, therefore, found necessary in the case of candidates for this department of Indian Service to inquire very carefully into their family history, and to reject those who show or inherit any neurotic tendencies.

The Importance of Good Digestion.

The chief cause, then, of breakdown in the tropics, apart from disease, being exhaustion — exhaustion of the nervous system, bringing with it its usual train of neurasthenic symptoms—it follows that the man who would maintain his health under these adverse conditions is one who has no difficulty in repairing waste. There must be no impediment to the rapid restoration of the exhausted system, whether from nerve strain or debilitating disease. In other words, his digestion and power of absorbing nutriment must be faultless. This is the first and most important condition for keeping good health in a hot climate, and without it there is no salvation. The man liable to dyspepsia, to "bilious" attacks, to diarrhœa in the summer months, to "gastro-enteritis" in any of its forms, and from trivial causes, had better stay at home. His difficulties of digestion and absorption will become intensified in the conditions we have considered, and he will, after a short residence in the tropics, "run down" in health, and in eight or ten years he will become one of His Majesty's bad bargains, always breaking down, frequently on sick leave, cursing the country, the day he crossed the Tropic of Cancer, and the doctors who passed him. These are the kind of men who furnish us, after a few years' service, with the most frequent and most difficult cases of neurasthenia—the neurasthenic with a dilated stomach. I have seen as many as three of these cases come before the India Office Board in a single afternoon. It would be well in every emaciated neurasthenic from India, to map out the stomach by percussion, as we do at that Board, and try for "splashing." Nearly all these cases will, on inquiry, be found to have been dyspeptic before leaving for India. I do not know whether the neurasthenia is the cause of the dilatation of the stomach, or whether the latter is a mere concomitant and aggravating condition of the neurasthenia, but their association in the same individual is a very frequent one, and leads me to conclude that in many maldigestion and consequent malnutrition determine the neurasthenia and emphasise its progress. A not

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improbable cause of this frequency of dilatation of the stomach among debilitated Anglo-Indians is their habit of drinking large quantities of fluids with meals.

In the absence of dyspeptic symptoms, or their concealment, how is a medical officer to detect a probable breakdown in this direction? Only by noticing how nutrition is being carried on under the favourable conditions of the temperate zone; and it is here that we find the value of the weight test. A good deal of not very good-natured chaff has been directed against the Army Medical Officer because he demands so many pounds of flesh, but failure to attain the weight normal to a given height and age is generally an indication of defective powers of digestion and nutrition, and is therefore an indication of liability to failure in the trying times of war, and the exhausting conditions of tropical life, in which so much of our soldiering is done. It is a rough test, but not one to be discarded, as was done in the latest Army orders. It is at least sufficient to put a medical officer on his guard against admitting men of faulty digestion, with all their tendency to break down.

But a faultless digestion is not only necessary to enable a man to repair readily the dilapidations of tropical life by healthy and rapid digestion of food, it is necessary also to enable him to kill, destroy, or render harmless the pathogenic organisms which he swallows with his food and drink. As is well known, the healthy gastric juice kills the cholera bacillus, and probably others. The comma-bacillus dies in an acid medium; so much so that some have said that to have an epidemic of cholera you must first have an epidemic of alkaline dyspepsia. I think they forget that the contents of the stomach are not always acid, and comma-bacilli entering the empty stomach in a draught of water would have a very fair chance of getting past the pylorus in a lively condition before the next meal. Nevertheless, I would back the man with a healthy digestion against the dyspeptic with a faulty one in the struggle with this fatal organism.

In my opinion, also, the man with a tendency to looseness of the bowels, no matter from what cause, is more liable to the evil chances of tropical life. It is not the constipated man who gets dysentery in the tropics, but the man with the opposite tendencies, whose mucous membrane seems to be in a more vulnerable condition. The same may probably also be said with regard to typhoid

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fever, though I have not sufficient experience in civil life to speak more positively.

The next question which I wish to discuss to-night is that of phthisis. In my report on the Congress on Tuberculosis at Berlin, in 1899, I went very fully into the question of the relative prevalence of tuberculosis in India, and I there pointed out that the native of India appears to have instinctively from immemorial time adopted all the dietetic habits best calculated to protect him against bovine tuberculosis. He boils all his milk, boils and converts all his butter into ghi, and he refuses to eat beef; he abstains also from alcohol, and the vast majority of people follow outdoor avocations. Whether his precautions against bovine tuberculosis are vain is not for me to decide; but from whatever cause it may be, he appears to succeed in escaping affection by the tubercular bacillus in quite a marked degree, especially in childhood and early life. But all native races do not escape equally, and the Dogra and the Ghurka, whose habits of life more closely resemble those of the European, are more subject than others to tubercular disease, especially of the cervical glands and lungs; and the European soldier and the prisoner in jail seem to be affected rather more than the corresponding classes in this country. There are no reliable statistics in India to enable us to compare the relative death-rate of the civil population with that of Europe in respect to phthisis, but such as they are they point to a comparative exemption from tubercular disease, and this is in accordance with the opinions of all medical officers who, like myself, have spent their whole time in India in civil appointments. With an experience of over 10 years in Calcutta, with an average of six to eight consultations a day, I do not think I saw more than half-a-dozen cases of pulmonary tuberculosis a year among well-to-do Hindoos, and in nearly every case over 40 years of age the patient was diabetic.

It is common enough, however, in the constitutionally weak half-caste, but is rare among Government officials of pure European blood. Chevers says that if a European "lands in Lower Bengal with perfectly healthy lungs, and lives well, prudently, and carefully . . . he will, residing there, never be attacked with phthisis, although he may be a member of a phthisical family," that is, although he may have a strong hereditary predisposition to phthisis. In this opinion most of us who have served in Lower Bengal will be nclined to agree, though I would substitute "rarely" for "never"

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in the above-quoted sentence. Those who have served in other parts of India may, however, have had a different experience, and it may be different with regard to European troops. How far this exemption may be due to precautions in the matter of boiling all the milk used, a precaution which the heat renders necessary, or how far it may be due to climatic causes, which diminish the liability to predisposing inflammations of the pulmonary mucous membrane, I will not pause to discuss, but I should certainly say that a strong hereditary predisposition to phthisis is no bar to residence in a climate such as that of Lower Bengal.

But what if the seeds of tubercular disease have already been sown in the lung? Should such a person proceed to a climate like that of Lower Bengal? To this question I could not give too emphatic a "No," and I entirely concur in Chevers's further opinion that "in that climate the course of the disease, once started, is exceedingly rapid, whether the patient be European, Eurasian, or native." In fact, I cannot recall a single case of arrest of phthisis in the whole of my experience in that part of India. The only case of the kind I can remember was that of a lady who left Calcutta and resided for four or five years in Simla. In her, a case of rather advanced phthisis, "a cure," in the sense of an arrest of all active manifestations of the disease, was effected, but only by change of climate.

Here, again, I only speak of the climate of Lower Bengal. I know of a case of active pulmonary disease in a strong man who came to Behar, a dry, hot climate, to follow the healthy out-door occupation of an indigo planter. and who completely recovered; but this, I fancy, was also somewhat exceptional. It is true that in India we follow all the indications of the open-air treatment; we live without doors or windows, day and night, and we have good cooks, and are well and copiously fed, and so far the conditions are favourable; but they are counterbalanced by the nervous exhaustion of which I have said so much, induced by the struggle to maintain a normal temperature, the loss of sleep during hot nights, the dust in which, in dry climates, we live and move and have our being, and by the debilitating effects of the exhausting diseases, which are the evil, but very frequent, chances of life in the tropics. It has been thought that the malarial parasite is inimical to the tubercle bacillus, but my own experience gives no colour to such a supposition. On the contrary, I think that the

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destruction of blood corpuscles, which is the great feature of the action of the parasite, and the exhaustion which that and frequent attacks of fever cause, are the reason why tubercular infection advances so rapidly, once it has started, in the hot, moist climate in which it was my fate to live.

Syphilis in its early stages can be treated as successfully in the tropics as at home; but it is not so with the advanced manifestations. Such cases should never be allowed to go to the tropics.

Diabetes, like albuminuria, runs a slower and milder course in the tropics than in temperate climates, especially after 40 years of age. This is especially true of natives, whose farinaceous and saccharine diet renders them very liable to the disease. Among them it is common among the very same classes who suffer from gout in this country, *i.e.*, the well-to-do, the sedentary, and those whose work is mental rather than bodily. Diabetes is, in fact, the gout of the Native Judicial Department. It is not common among Europeans living in the tropics, and after all the early attempts to cure a case of diabetes by diet, &c., have failed and are given up, such a case would probably do just as well, if not better, in the tropics than if he stayed at home.

REMOTE EFFECTS OF TROPICAL LIFE ON EUROPEANS.

By JOHN ANDERSON, C.I.E., M.D., F.R.C.P.

MR. PRESIDENT AND GENTLEMEN,—In speaking to you to-night of the remote effects on Europeans of residence in tropical and sub-tropical climates, I shall refer more particularly to the diseases of India, though time will only allow me to deal with some of them. I fear you will regard the greater part of what I am going to say as truisms; at the same time by connecting the cause with the effect, it may be possible to give the latter some practical interest.

Many diseases have been attributed to heat which are more justly due to defects in personal hygiene and habits of life, to an over-stimulating diet, to a too liberal use of alcohol, and to an over-indulgence in tobacco; moderation and self-restraint in such matters are necessary for the maintenance of health in warm climates, for

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it must not be forgotten that the resident has to adapt himself to the heat environment, as it cannot possibly be adapted to him.

Modern bacteriological research has given greater precision of view to the problems of the cause and prevention of disease than has hitherto existed in the world's history. Possibly we are approaching a time when we shall recognise that acclimatisation has but little to do with the question of disease, beyond the fact that varying degrees of heat and moisture afford more or less favourable conditions for the development of the disease germ, and perhaps for the more rapid progress of the malady to which its presence gives rise.

It, nevertheless, goes without saying that, for climatic reasons, residence in hot countries increases the risk to life and health in immigrants from colder climates.

The physiological changes that are induced by heat are numerous, and worthy of passing notice. The body-temperature is raised nearly half a degree. The respiration is slower and the capacity of the chest for air is greater because the temperature of the atmosphere determines a greater blood-supply to the surface of the body for the regulation of its temperature; thus the relative proportion of blood and air in the lungs is altered. The pulse is slower, the renal secretion is reduced, while that of the skin is much increased. The tone of the nervous system, including the sympathetic, is depressed, so that the visceral, as well as the general circulation, is influenced. Digestion is slower, and nutrition is less active. These may be regarded as the physiological disadvantages of residence in a hot climate: but they are encountered, when the individual is young and is able, possibly without any great stress, to adapt himself to them. When a man returns to Europe after long residence, say in India, he changes back to the conditions of his early life with much more difficulty, for he is constitutionally less elastic and less adaptable to the second change than he was to the first. The habits of Indian life have become natural to him, and we have to add to the loss of youth the pathological changes which are the sequelæ of tropical diseases. The sudden removal from a life of interesting activity to one of idleness, to say nothing of the diminished pecuniary resources due to the differences between full and retired pay, tend to bring about a degree of mental depression which does not assist the second process of climatic adaptation.

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The effects of tropical life upon the European are to be measured, not only by the sickness and death-rate that occur during residence in hot countries, but also by the state of health and the length of life that follow the return to a temperate climate, as compared with those of the same age who have remained in Europe.

The following statement, for which I am indebted to the Standard Life Assurance Company, shows that the comparison is unfavourable to those who have lived long in hot countries. The Secretary of the Standard Company writes me as follows:—

"Some years ago we took out the mortality of policy-holders who had been in India, but were then living retired in this country. The data on which the results were based were not extensive, but I remember the results, as far as they went, were unfavourable; the deaths being something in the ratio of 110 to 100 expected by the English life table, whereas the ordinary home mortality amongst our policy-holders is only about 85 per cent. of the table. The result indicates that our Indian 'extra' must not only cover residence *there*, but also the increased mortality after return to this country when only home rates are payable."

It is not questioned that malaria stands prominently forward as the active agent for evil upon the health and constitution of European residents in hot countries. I need hardly enumerate the diseases that directly owe their origin to malaria, and are occasionally met with long after the sufferer has returned to Europe. These deferred manifestations of malaria are often of a very irregular type, and symptoms frequently yield surprisingly to treatment which, if regarded only from a clinical standpoint, and without a knowledge of the antecedents and medical history of the patient, might lead to an unduly grave diagnosis, and cause needless anxiety.

To illustrate my meaning: albuminuria is a condition which is often present in those who have lived in hot countries. Sometimes this is accompanied by extensive œdema of the lower extremities, and is suggestive of Bright's disease in an advanced stage. The microscope will not, however, reveal the presence of renal casts, and the disappearance of the symptoms under the influence of quinine, iron, and arsenic, is often as unexpected as it is gratifying. It is scarcely necessary to add that the various forms of malarial fever, or their recurrences, frequently come under notice, and I do not propose to do more than refer to them.

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I have seen cases of possible but doubtful malarial origin, with an undulating temperature line ranging from 99° to 101° F., which, while presenting no visceral complications, had the usual febrile accompaniments of anorexia, lassitude, and debility, to which marked anæmia was added, but had no specific symptoms to indicate their exact nature and origin, or to lead to the establishment of a correct diagnosis. Such cases obstinately resist treatment, and end as mysteriously as they begin, drugs appearing to have no effect upon them. After an indefinite time the fever "stops," and the patient slowly recovers.

Of malarial neuralgiæ, the most common are facial and hemicranial, brow ague, and intermittent headache, but sciatica, intercostal and abdominal pain, burning of the feet, and acute tenderness of the shins, are often attributable to the same cause.

The length of time that malaria sometimes remains latent is remarkable; of this, I may mention a striking instance that occurred some years ago in my own experience. A gentleman consulted me for frontal headache of extreme severity. He had taken many remedies and obtained no relief. On questioning him I found that this severe pain came on about 4 o'clock in the afternoon, and had done so for many days. This periodicity led me to look for a malarial history, and I learned that my patient had been in Asia Minor 40 years before, and that while there he suffered severely from malarial fever, on account of which he came home. He then recovered, and had not since suffered from fever or been in a malarial climate. As I was consulted about noon, and the attack of neuralgia was due in four hours, I at once prescribed 10 grains of quinine, with the happy result that the pain did not appear on that day. Smaller doses of quinine, combined with arsenic, restored the patient to health.

Here, *par parenthèse*, I may mention the interesting and noteworthy fact that locomotor ataxy is seldom or never met with in the natives of India. Europeans, however, do so suffer, and since natives suffer much from syphilis their practical immunity from locomotor ataxy leads to a possibly unorthodox doubt as to the correctness of the theory of the necessarily syphilitic origin of this disease.

I remember the case of an officer who returned from India, and whose symptoms very closely resembled ataxia, so much so that an eminent neurologist agreed with me in so diagnosing the case. This

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opinion was supported by the fact that the patient had suffered from syphilis, but in the course of time he made a perfect recovery, has since become the father of healthy children, and is now, I believe, quite well. How far his symptoms really depended upon the specific cause to which they are usually attributed, and how far to a condition of malaria, I cannot say, but his perfect recovery is an equally satisfactory and interesting feature in his history.

Serious conditions of the nervous system often result from sun or heat stroke. In grave cases, when the initial seizure is survived, such sequelæ as epilepsy, blindness, deafness, and even dementia, sometimes occur; affections of the motor nerves causing paresis or paralysis are much less frequently met with. In milder instances the patient suffers, perhaps for many months, from inability to concentrate thought, or to maintain prolonged mental effort. He cannot bear any exposure to the sun, even in cold climates, and heated, ill-ventilated, or crowded rooms induce headache, frontal or occipital. These cases suffer also from irregularity of the heart's action, insomnia, irritability of temper, depression of spirits, a morbidly nervous fear of every-day occurrences, such as riding, driving, or a railway journey, and have other similar evidences of neurasthenia and disturbed nerve function. These symptoms often disappear entirely if the sufferer can remain for a sufficiently long period in a temperate and cool climate, if he is careful to avoid exposure to the sun, to eschew alcohol, and to avoid all forms of excitement.

Another and more remote effect of tropical disease is an apyrexial cachexia which is marked by a progressive anæmia so severe that it closely resembles the pernicious form of blood degeneration. The patient looks cadaveric, his spleen is enlarged, he suffers from intermittent neuralgia, possibly anasarca, and his debility is extreme. The crescent-shaped plasmodium may be present in his blood, yet he may have suffered little, if at all, from malarial fever. The physician is consequently liable to be put off the track, and the malarial origin of the case not being recognised or treated, the patient's symptoms probably increase in gravity and may end fatally. In these cases of cachexia and progressive anæmia, the quantity of the blood is seriously diminished, and its quality gravely affected. The corpuscles, both red and white, are reduced in number, and the red are altered in colour, shape, and size; in severe cases the plasma is impoverished, and there is a tendency to visceral infarction, and

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to the formation of coagula in the large vessels and cardiac cavities. There is also a liability to hæmorrhages from mucous membranes; the gums are not infrequently swollen and spongy and bleed on the slightest provocation. Sometimes this condition goes on to suppuration, and pus wells up between the gums and teeth. Such a state of the gums much resembles that met with in scurvy with which it is, however, in no way necessarily connected.

A serious and common effect of malaria is a chronic hypertrophy of the spleen. I have seen the enlargement extend considerably below the level of the umbilicus in varying degrees of induration.

Of the diseases which malaria complicates the list is long. Of these, that of typhoid fever is perhaps the most grave, often leading to a critical condition which is not indicated by the typhoid fever alone. For my own part I do not believe that the so-called typho-malarial fever has any separate existence or that it is, as some seem to consider, a hybrid product or a separate and distinct variety. I believe it to be veritably and indeed typhoid fever, complicated by the presence of previously acquired malaria.

Hepatic disease of malarial origin usually commences with enlargement, which sometimes becomes chronic, and frequently tends to end in cirrhosis. The ascites, not uncommon in splenic enlargement, is probably largely due to hepatic obstruction.

As a remote effect of Indian life, I must mention abscess of the liver, because it often does not show any evidence of its existence until years after the patient has returned to Europe. Such cases are very puzzling. Occasionally the presence of an abscess is suspected, although in the absence of all positive evidence, and in spite of most careful examination, it cannot be located or diagnosed. I have more than once seen an abscess so masked by an apparently malarial temperature, the absence of enlargement, tenderness, and other physical signs, that an error in diagnosis resulted. Repeated recurrences of illness with marked febrile symptoms, and a very oscillating temperature, alternating with intervals of health, have much impressed me with the need that exists in such cases for the exercise of great caution in diagnosing them. One is put very much on the alert if the fever does not yield to quinine, if the plasmodium malariæ cannot be found in the blood, and if there is a history of dysentery.

I remember a case of hepatic abscess that occurred in an artillery officer eight years after his return from India. He had suffered

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severely from dysentery while there and also from malarial fever. He had several relapses of dysentery after his return home and also attacks of fever which strongly resembled an irregular form of malaria. During these eight years he had many long intervals of good health. The attacks of dysentery, or dysenteric diarrhœa, usually yielded readily to treatment; the febrile attacks did not, they ended, as they began, "of themselves," unaffected by any remedy. This officer was seen by several physicians of tropical experience, each one of whom was haunted by the suspicion that there might be an abscess in his liver; they sought often and with care, but in vain, for confirmation of their fears. Eight years after his return to England, an abscess in the liver opened into the lung. This case illustrates the difficulty sometimes met with in diagnosing hepatic abscess and of distinguishing its febrile phenomena from those attributable to some forms of malarial or climatic fevers.

Although the geographical distribution of dysentery is not limited to any climate, in its chronic form it is frequently a remote effect of long residence in hot and malarious countries. Not that dysentery is ætiologically dependent on malaria, but because those who suffer from malarial cachexia or anæmia are more liable than others to suffer from it in its acute form, and in them particularly relapses of chronic dysentery recur often with considerable frequency. Chronic dysentery is, as we all know, a very intractable malady, and is very liable to relapse after long intervals of good health. Such are to be regarded as recrudescences of an acute or sub-acute degree, and they occur on slight provocation.

In an official report just published by the India Office I find the following statistics:—

In European soldiers in 1899 the admissions per 1,000 from dysentery were 25, and the deaths per 1,000 were ·65, while in native troops the admissions per 1,000 caused by dysentery were 38, and the deaths ·35. In the same year the mortality from abscess of the liver was 32 times greater in European than in native soldiers. These official figures for 1899 are consistent with those of several former years. They strike me as being very interesting and not a little perplexing. If an attack of dysentery is the most common precursory cause of liver abscess, how is it that the native soldier should be so much more liable to dysentery, and yet that liver abscess should occur so relatively seldom in his case, as compared with its occurrence in Europeans? Can the difference

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be accounted for by the diet and habits of the respective races? The natives of India are not, as a rule, meat eaters, nor consumers of alcohol in any form. It has been suggested with regard to the comparative immunity of the natives of India from typhoid fever, as compared with its marked prevalence among Europeans there resident, that it may in like manner be explained. The natives of Nepaul, from whom our Ghoorka regiments are recruited, are the sole exception to this immunity, and they alone of the native races drink alcohol and eat animal food. These are very suggestive facts.

An intestinal disease of great interest is the ætiologically obscure sprue or psilosis, called by some a chronic tropical diarrhœa, but differing pathologically from that affection. Whatever this disease may be ætiologically, it markedly possesses a mysterious element of latency. As far as I am aware, no person has been known to suffer from it who has not lived for some time in a hot climate, but certain it is that those who have returned from such climates and have been for some years in Europe may, and do, so suffer.

I have recently had under my care a general officer who has not been in any tropical country since 1880. Prior to that he was for some five or six years in India, and was always stationed in the hills in the hot weather, being in the plains only during the cold seasons. About two years ago he began to suffer, for the first time, from a very irregular form of diarrhœa, and from this the symptoms developed until they assumed the characteristics of psilosis. The patient is advanced in years, and in consequence the prognosis, in his case, is grave, although in the meantime he is, and has been for the last two months, very much better. His own belief is, that his present improvement is due to a free indulgence in strawberries last summer, and when strawberries were no longer available he turned to mulberries, which he continued to eat as long as they were obtainable.

Another case, that during the last few months I have seen several times in consultation, is that of a lady who lived for many years in India, and with the exception of occasional attacks of malarial fever, enjoyed good health in that country. She returned to England 15 or 16 years ago, and since that time has resided in the south of England and the Channel Islands. In May last, after a time of great trouble and anxiety, owing to the serious illness of a member of her family, she developed unmistakable symptoms of this disease, and she is still seriously ill. It is to be

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noticed that neither of these cases suffered from this disease or any intestinal affection while in India.

I am quite unable to offer any explanation or give any reason why this disease should be confined to those who have resided for a considerable time in tropical or sub-tropical climates, and still less am I able to account for the lapse of time that in some cases occurs after the return of such persons to a temperate climate before they have any manifestation of the disease. The tolerance of fruit in these cases is noteworthy; in my experience it is usually well borne, and in patients who cannot, or will not, take sufficient milk, I have found the addition of fruit to the diet of service. I learnt this from reading a paper or letter in one of the medical journals, which I think was written by Dr. Thin.

I must refer briefly to those protracted forms of Mediterranean fever which are attributed to the presence of the micrococcus melitensis; an organism discovered by Surgeon-Major David Bruce in 1887. In these fevers the temperature may be said to constitute the disease, though rheumatism and neuralgia are frequent concomitants and sequelæ. The sufferers come home; neither quinine nor any other drug has any effect on their symptoms, but almost all of them eventually recover, the mortality being about 2 per cent. The fever is not in any way associated with malaria or typhoid, but in its early stage may be easily mistaken for the latter. Later it is diagnosed without difficulty. The temperature is undulating, and after several weeks' duration an interval of some days often occurs, to be succeeded by another period of fever. This order of events may be many times repeated, and the duration of the illness may extend over many months. I recently saw a case that lasted 14 months, of which nine were passed in this country. The spleen is enlarged, and that is the only feature of pathological anatomy in evidence.

I see here to-night several physicians who have experience of tropical diseases. It will be interesting, and I believe it will be instructive to many if they can throw light on the questions I am unable to answer, and to which I have ventured to invite your attention.

The PRESIDENT expressed the Society's thanks to Dr. Crombie and Dr. Anderson for their exceedingly interesting papers, in which the authors had departed considerably from the ordinary beaten track. He said one could not help being struck, in listening to the papers, by the

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enormous advance that must have been made in the study of tropical diseases, which, by those who were not practically familiar with them, had been hitherto regarded as something altogether different to what they were accustomed to deal with in this country. But the papers enabled them to appreciate the importance of individual fitness to enter upon tropical life, with special regard to the adoptive and resistive capacity.

Dr. THIN said he would not attempt to follow the theoretical considerations which Dr. Crombie had put forward, but paid a tribute to that gentleman for putting the question on such a scientific basis. That aspect of the subject required more consideration than had been possible during the reading of the paper. He would simply refer to matters of common observation which had come into his own experience, and which bore upon the doctrines Dr. Crombie laid down. He claimed to do that because, in more than 20 years' experience, he had found that if a young man selected for tropical work showed any sign of weakness or habitual bad digestion, or was of insufficient weight, more especially if he was tall and slender, he was almost certain to break down in some form or other within 10 years. A number of years ago there was an institution represented in this country which had its men medically examined before they were sent out. When a man was certified sound and healthy, the observations made about his not coming up to the requirements of physique were apt to be waived. The head of that institution received in one day four telegrams for fresh men to be sent out to various agencies, to replace four who had broken down in health. On referring to the records, it was found that in each case it had been originally pointed out that the candidate was not in that robust and energetic health which was advisable for service in the tropics. The result was the insistence on more stringent instructions, and greater care in the selection of men; and since then there had been much less sickness amongst the assistants.

With regard to the effects of malaria in this country in persons who had been ill of that disease in the tropics, the symptoms shown here were very obscure, and deserved much closer study than they had received. Since the discovery of the malarial parasite, he had conducted a careful investigation on a consecutive series of cases of so-called malarial fever occurring after four or five years' residence at home. In only one of those cases, namely, one from Jamaica, did he find anything suggestive of the malarial parasite, and in that case the only suspicious appearance was a solitary stained body in a red corpuscle, with no pigment. He was satisfied that many cases had been treated in this country as active malaria in which the poison had ceased to exist. How could one account for those febrile symptoms? In many cases it was impossible to find out the cause. Such people would have an attack of fever which got worse on the second day, and in three or four days would die away. Quinine did no good in such cases, but rest in bed, with fluid food and no drugs, resulted in a disappearance of the fever. There was no pigment in the white blood corpuscles, and no parasite could be found in the red corpuscles. The heresy that every man who had been in the tropics must have malarial fever was bound to die, and the sooner it died the better. A friend of his who died two or three summers ago in London of malignant disease believed he had malaria, and his own diagnosis was borne out by other men whom he saw. He, Dr. Thin, examined the blood several times and concluded there was no malarial fever, and begged him to give up the quinine which he had been taking. He

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would not do so, but took, with the concurrence of one of his medical advisers, a three weeks' course of 20 grains of quinine per day. The real nature of the disease soon became manifest, and then the futility of the quinine treatment was seen. He remembered another case—that of a man who had returned from India, and was ill with feverish attacks at one of the towns on the south coast. The diagnosis of malaria was made, and quinine was administered. He returned to town, and got rid of his fever by lying in bed a few days. A few months afterwards he died of acute congestion of the kidneys, and *post-mortem* examination showed large dysenteric ulcers in the lower bowel. Another case was that of a patient who was suffering from well-developed psilosis. He had got well under careful treatment, but did not continue with it, and relapsed. He then consulted, in London, a physician whose knowledge of intestinal and stomachic ailments was well known. But because the man had been in the tropics he was put on a course of quinine for three months, which, of course, did his diarrhœa and other symptoms no good; indeed, it possibly did them harm.

With regard to the cases in which there were symptoms of psilosis long after a return from the East, they were very remarkable. A lady in the north of London a few years ago was taken ill with a sore tongue, intestinal irritation, emaciation, and debility. The medical man who attended her concluded that the disease was one of which he had never seen an example, and told the patient so. In forming his opinion he was not influenced by knowing that the patient had been in the tropics. He (Dr. Thin) happened to see the case afterwards, and found it was more than 18 years since the patient had left the south of China. During those years she had had fair health, and the symptoms of the complaint referred to did not develop until that time. The case proved fatal. Such cases were not very common. He particularly wished to record his thanks to Dr. Crombie and Dr. Anderson for their remarkably thoughtful papers, and recommended the Fellows to study them carefully after they were printed.

Mr. Cantlie said he agreed with all that had been said by Dr. Thin with regard to sprue. He was particularly interested in the remarks made by Dr. Anderson on liver abscess, and the statistics which had been adduced to show that the number of European soldiers in India who suffered from dysentery was very small, but the number of those who contracted liver abscess was very large. Mr. Cantlie's contention was that dysentery was not the cause of one particular variety of liver abscess, the one which he called supra-hepatic abscess, an abscess which occurred above the liver and beneath the diaphragm, and had little to do with liver ailments, and nothing to do with dysentery. In many of those cases dysentery was not present. He knew of one case which arrived in Hong Kong, and developed supra-hepatic abscess pure and simple within four or five weeks of arrival. Another case was that of a young man of 30, a captain of one of the cruisers, who developed liver abscess within three months, though he had never had dysentery. He thought those abscesses were determined by chill, and just as pneumonia resulted from "catching cold" in England, so did hepatic troubles in the tropics.

The question of rest in malarial fever had been raised by Dr. Anderson and by Dr. Thin. He knew of one case in which a lady had left the tropics two years, and had a constant temperature of 99° to 101°. No drug did any good, and benefit was obtained only by rest in bed. Occasionally there were accessions of fever, then the spleen became

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enlarged, and as speedily subsided after a few days' rest in bed. During these splenic enlargements and attacks of high fever evidence of malarial infection of the blood was sought for, but with negative results.

Dr. LAWRIE thought diseases were sometimes attributed to tropical climates, for instance, that of India, which really, as Dr. Anderson had pointed out, were due to errors in diet. Eight or ten years ago a well-known cricket team went to India to play against the Parsees at Bombay. He went to Madras to see them play, and, to his surprise, he found that those splendid English cricketers were easily beaten. The reason given was that they had not become acclimatised, and were suffering from the effects of the sun. He travelled back to Hyderabad with some of the members of that British team, and at a station *en route* he dined with them. He noticed that they ate enormous quantities of curry, and drank considerably of champagne. He found that they had been in the habit of eating three hearty meals a day. At a big dinner at Hyderabad, he sat next to the captain, who was disturbed about the results of his matches, and asked him, Dr. Lawrie, what ought to be done to enable them to beat the Parsees. His reply was, "Make the team teetotallers." That was done, the effects of the sun disappeared, and the Parsees were beaten.

Dr. ANDREW DUNCAN said, with regard to tubercular disease amongst Europeans in India, that the effect of it differed with the part of India concerned. In Bombay he once saw an officer who had well-marked commencing tuberculisation of the apex of the left lung. He was losing flesh, he spat blood, and there were some sub-crepitant rhonchi. He advised him to go back to England, but this his circumstances would not permit. Seventeen years afterwards he heard that the officer was practically all right. In the Punjab tubercle presented a different feature, and he had not known a case there which did not quickly die. The most rapidly-fatal cases of phthisis he had seen were among the Goorkhas, where, two months after the first signs of tubercle were found, the patient would generally be dead of the disease. The same thing was seen in the case of Europeans in the Punjab, although the course was not quite so rapid.

With regard to bowel affections, he thought Europeans who had once had dysentery should be very careful about going out to India again. A man in the Burma Police was invalided home for dysentery and recovered; then, much against his will, he had to go out again. A fresh attack followed, and he was finally sent home.

With regard to sunstroke, Dr. Duncan thought persons who had been affected should not be again passed for service abroad.

With regard to syphilis, he thought a man who had once had that disease should not go to India. In 1898, when the Royal Irish Fusiliers landed in Egypt from Burma, they had to be sent home at once, as the whole regiment was unfit from that disease. In Chitral, out of 5,822 men, 462, or 8 per cent., had to be rejected on account of syphilis. After the regiment moved up, another 4½ per cent. had to be sent down to the base. Five years ago the admissions on account of syphilis in India were equal to the entire loss of three regiments for the whole year. In 1893, 1,500,000 days of duty were lost to the public services from the disease among soldiers in India; and at that rate the whole of the British army in India would have been treated for syphilis in two years. He alluded to a striking instance of the acquisition of syphilis by a very large number

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of Goorkhas from a woman who had a hut only 10 yards outside the cantonments. As she was outside the limit no one could interfere with her. He thought people of so-called nervous temperament should not go to India; all the men of that kind he had known who had gone out had been invalided home.

With regard to age, in Earl Roberts' famous march from Cabul to Kandahar, all the Tommies kept well except a certain number who belonged to a new draft of young soldiers in the 72nd Highlanders. There was no doubt that a man was not fit for tropical life before 25 years of age. The Army Medical Regulations required him to be 20, but that was constantly evaded. Earl Roberts agreed that the minimum age for the tropics should be 25.

Dr. Crombie said but few remarks were called for from him in reply, as the speeches had been chiefly of a confirmatory character. With regard to sunstroke, that was mentioned in his paper, but from want of time was not read. Explaining a chart which he exhibited, he pointed out that heat apoplexy was most likely to occur when the nervous control was very much exhausted by heat, and then, at the time of the normal evening rise, and especially if this normal rise were exaggerated by exertion, or the nervous system disturbed by excessive or chronic alcoholism, the nervous control gives way, and the body temperature, no longer inhibited, may rise to 107° or 109° F. This is probably the true explanation of heat apoplexy, which is most frequent in the evening.

Dr. Anderson said Dr. Thin had practically confirmed his own observations in the paper. With regard to liver abscess, he thought no one would doubt that abscess in the liver might occur without any relation to dysentery. He (Dr. Anderson) had referred to dysentery as a *common* precursory cause. Diet and temperate habits were very important in those who were intended for a tropical career. With regard to Dr. Duncan's opinion that dysentery cases should never be allowed to return to India, Dr. Anderson thought that was a rather severe sentence upon a young man who had adopted the Civil Service or a military career in India. A great deal depended upon the severity or degree of the dysentery, and upon the length of time that he could remain in a European climate. Time not unfrequently effected a permanent cure. He agreed in thinking that the Medical Boards were hardly liberal enough in giving sufficient leave in dysentery cases. He doubted whether Army Regulations allowed Medical Boards to do more than they do.

With regard to syphilitic people not going to India again, he could not agree. He had often seen syphilis successfully treated in India. Did not that also depend on the degree of the syphilis and the extent to which it had been treated? He, however, agreed with the reference which had been made to the inclusion of syphilis in the Contagious Diseases Act.

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January 13th, 1902.

OBSERVATIONS ON THE SURGICAL TREATMENT OF OBSTRUCTIVE JAUNDICE FROM AN EXPERIENCE OF OVER 200 CASES.

By A. W. Mayo Robson, F.R.C.S.

My recent operative experience in cases of chronic obstructive jaundice, especially in those associated with gall stones and with chronic pancreatitis, has led me to take a more hopeful attitude in considering the treatment of deeply jaundiced patients from a surgical standpoint; and it is with a view to the expression of my views that I have ventured to respond to the kind invitation of the Council of the Medical Society by giving this paper to-night.

In expressing an opinion that the surgeon has, as a rule, no business with these cases until the symptoms are well marked, I think it should also be distinctly recognised, not only that no patient should be allowed to die of obstructive jaundice without the question of surgical interference being considered, but also that the question of surgical treatment should be raised at a time when operation may be undertaken, if called for, with a reasonable probability of success.

It has been my unfortunate experience to have to give an opinion on a very large number of such cases at a time when any active interference was out of the question, and when the only interest in the case was the question of diagnosis; and this must have been the experience of many of my surgical brethren.

Diagnosis.

In all chronic jaundice cases the first and often the most difficult question is that of diagnosis, for if we can come to a definite conclusion regarding the cause of the obstruction, the proper treatment to adopt can usually be readily decided on.

The following causes must be taken into consideration :—

1. Common duct cholelithiasis.
2. Chronic pancreatitis.
3. Simple stricture of the common bile duct.

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4. Inflammatory adhesions causing pressure on or stenosis of the hepatic or of the common bile ducts.

5. Hydatid disease of the liver pressing on or discharging into the bile ducts.

6. Gummata implicating the ducts.

7. Chronic catarrh of the bile ducts.

8. Cancer of the common bile duct.

9. Cancer of the head of the pancreas.

10. Cancer of the liver associated with jaundice, and either due to catarrh or pressure.

11. Cirrhosis of liver.

12. Other rare causes, such as aneurysm of the hepatic artery or of the aorta, and other tumours of the liver, gall bladder, pylorus, kidney, or intestine pressing on or occluding the common bile duct.

It will be at once seen that surgery holds out a good prospect of cure in the first five causes enumerated; that medical treatment alone is advisable for causes 6 and 7; and that in the remainder, with certain exceptions, relief only can be hoped for either from medical or surgical treatment.

Although one could easily spend an hour or more in discussing the diagnosis of the various causes, I think it will be more advantageous if I simply mention one or two points that I have found useful in guiding me to a conclusion as to whether the case is suitable for an exploratory operation or not.

A painless onset of chronic jaundice must always give rise to the suspicion either of chronic catarrh dependent on cancer of the liver or of occlusion of the hepatic or common bile duct by growth, and, if this be associated with distension of the gall bladder and rapid loss of weight and strength, cancer of the head of the pancreas will probably be found.

On the other hand, the history of an attack of pain in the upper abdomen, followed within 24 or 36 hours by jaundice, and preceded by so-called "spasms," either recently or at some remote period, is strongly suggestive of cholelithiasis. In the latter case the jaundice will probably be less intense than in the former, and an intermittent fever with chills and sweats will probably either be present or have been noticed at some stage of the illness.

Enlargement of the liver is much more common in obstruction due to cancer than in that from gall stones, though it may be

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present in either; in cancer, however, nodules or irregular bosses may be discovered, and in gall stones an elongation of the right lobe of the liver, which is apt to be mistaken for a distended gall bladder, can often be felt.

The presence of ascites is suggestive of malignant disease, but in one case on which I operated, although there was a fair amount of fluid in the abdomen, I could find no sign of cancer, though there was cirrhosis of the liver due to biliary stagnation from the presence of a gall stone in the common duct for three years. I therefore persevered with the operation, and, after removing the obstruction, stitched up the omentum to the parietes in order to establish a collateral circulation. The patient, a man of 56, is now well. This is, however, an exceptional case, and, as a rule, the presence of ascites negatives any radical operation on the bile ducts.

In the early stages difficulties will arise that time will clear up: for instance, the jaundice of gall stones is rarely continuously the same, but increases or diminishes from time to time; whereas the jaundice of obstruction due to growth steadily increases or tends to become absolute, especially in cancer of the bile duct or the head of the pancreas.

The element of time alone is of importance in diagnosis, for jaundice with malignant disease runs a very short course, whereas jaundice from gall stones or some simple cause may go on for several years.

It must not be forgotten that gall stones and cancer frequently coexist, and in the case of cancer of the gall bladder gall stones are nearly always present, and the combined disease may form a tumour before the onset of jaundice, though some degree of jaundice usually supervenes early in the disease.

Fat in the motions and glycosuria, with very rapid wasting, are suggestive of pancreatic trouble.

Ague-like symptoms more frequently accompany gall stones in the common duct than malignant disease of the ducts or of the head of the pancreas, but several cases that have come under my notice show that this rule is subject to exceptions.

After the abdomen is opened the discovery of adhesions in the neighbourhood of the gall bladder, and a contracted gall bladder, are strongly suggestive of gall stones, though I have found ulcer of the pylorus producing adhesions to the gall bladder, and at the

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same time setting up pancreatitis, and then, by pressure of the swollen pancreas on the common duct, setting up jaundice.

If no gall stones be found, but the head of the pancreas be swollen and harder than normal, the surgeon should not too hastily condemn the patient as the subject of cancer of the head of the pancreas, for the swelling may be a chronic pancreatitis, and may be curable by cholecystotomy, as I have proved in a considerable number of cases. Nor does the presence of enlarged glands warrant a gloomy prognosis, for it is quite common to find discrete glands in common-duct cholelithiasis and in chronic pancreatitis, though if the glands be fused together and infiltrating the neighbouring tissues, the outlook is far from good.

A rigid right rectus abdominis and tenderness one inch above and to the right of the umbilicus, is as suggestive of gall stone trouble as is McBurney's tender point of appendicitis.

The appearance and feel of a tumour composed of matted omentum and the viscera adjacent to an inflamed gall bladder are often very deceptive, and in two at least of my earlier cases, after exploring such a mass with a hollow needle, I closed the abdomen and gave a gloomy prognosis, which was proved to be wrong by the patients steadily improving and completely recovering. Before seriously interfering with the adhesions, and thus being committed to the following out of a dangerous and futile operation, it is desirable to raise the abdominal wall and carefully examine the liver and adjoining parts so as to be assured of the absence of secondary nodules of cancer.

If in doubt, it is better carefully to detach the omentum covering the gall bladder itself, when it may fortunately turn out that the viscus is full of gall stones and pus, as in cases 56 and 57 to be mentioned later, in both of which inoperable cancer had been diagnosed and a gloomy prognosis given, but in which cure by operation was effected, and the patients are now well; or even if, as often happens in this region, cancer be discovered and proves to be local, a radical operation may be undertaken with good prospect of success, as in a case where, three years ago, I removed from a middle-aged gentleman a wedge of liver and the gall bladder affected with carcinoma, and he remains in excellent health to-day; and in another case, where, in a woman aged 56, I excised the gall bladder, the pylorus, and a portion of the liver, the patient being now, well on in the second year after operation, in good health.

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If, however, secondary nodules be seen or felt in the liver or omentum, the diagnosis of irremovable disease will be at once arrived at, and the abdomen closed without delay.

A hæmorrhagic condition is common in cases of chronic jaundice, especially if due to cancer of the head of the pancreas or to interstitial pancreatitis. If not excessive, this can be corrected by chloride of calcium, or possibly suprarenal extract of gelatine; but, if severe, it will undoubtedly seriously add to the risks of operation.

If a patient at or past middle age has heart disease or albuminuria, operation should not be lightly entertained, and such cases have, as a rule, to be content with the relief that can be given by medical treatment.

Treatment.

The second question, as to treatment, depends on the diagnosis:—

1. If the diagnosis be doubtful an exploratory operation is advisable, providing the general condition of the patient renders it probable that such a procedure *per se* apart from what is to follow will not hasten death.

2. If malignant disease be positively diagnosed, operation can, with some few exceptions where it is possible to completely remove it, do little good, or at the best can probably only prolong life for a short time.

3. If gall stones, or, in fact, any of the first five enumerated causes be diagnosed, operation is decidedly advisable if the patient be at all in a condition to bear it.

1. *When the Diagnosis is Doubtful.*

It will be convenient to consider these three conditions *seriatim*, and first of all the question of operating for the purpose of clearing up the diagnosis, with a view to at once closing the abdomen if unfavourable conditions be found, or, if advisable, performing whatever operation may be necessary at the time in order to bring about relief or attempt a cure.

The uncertainty as to the cause of the obstruction in many cases of jaundice is what makes this class of cases of such extreme interest, both to the physician and surgeon; and although ripe experience will frequently enable a diagnosis to be arrived at by instinct as it were, yet mistakes will be made at times, and patients

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condemned when prolongation of life, or subsequent cure by operation proves that the most skilful diagnostician may err.

The following brief abstract of cases condemned as hopeless, but afterwards operated on, demonstrates my meaning better than a multiplicity of words. The numbers, where present, refer to the order of the cases in my notebook:—

No. 56.—Woman, aged 50, residing at Burnley. Jaundiced nine months; great loss of weight; pain not a prominent symptom, but present. Pronounced to be cancer by an eminent Manchester physician, and operation not advised. Operation, September 29th, 1892. Gall stones found in gall bladder and common duct. Cholecystotomy and cholelithotrity performed. Good recovery. Quite well when heard of a year later.

No. 57.—Woman, aged 50. Jaundiced 10 months. Pain with ague-like attacks. Pronounced malignant by a distinguished physician in Sheffield, and operation deprecated. Operation, October 4th, 1892. Gall stones removed from gall bladder and common duct. Good recovery; well in 1893.

No. 66.—Woman, aged 54. Sent to me for tumour in gall bladder region, associated with pain and jaundice. Gall stones diagnosed, but on opening the abdomen a hard nodular tumour discovered, yielding only blood to exploring syringe. Thought to be malignant, and abdomen closed. Complete and perfect recovery; and when seen some months later all trace of tumour had disappeared.

No. 213.—Man, aged 45. Painful epigastric attacks for 12 months, with vomiting. Deep jaundice for three months, with ague-like attacks. Loss of 2½ stones in weight. Distended gall bladder. Operation, March 29th, 1898. No gall stones. Tumour of head of pancreas discovered with thickening of common duct, thought to be cancer. Drainage of gall bladder for 10 days. Complete recovery. Quite well at present. Case, chronic pancreatitis.

No. 233.—Man, aged 39. Gall stones, seizures for five years, and discovery of concretions in motions. Operation, September 22nd, 1898. Gall bladder and liver infiltrated with what appeared to be cancer, though nodules not large enough to be felt through abdominal walls. Some gall stones felt deeply in bile passages. Thought to be useless to attempt complete operation. Patient recovered, lost his jaundice, gained weight, had no further recurrence of pain, and was reported to be quite well May 24th, 1899. This case I cannot explain, as there was every appearance of cancer of the liver.

No. .—Man, aged 51, said by eminent London physicians to be suffering from cancer of liver and to be incurable. Jaundice intense for nine months; great loss of flesh: very little pain, and no ague-like attacks. The only suggestive symptoms were the history of intermittent pains in the upper abdomen some years previously, and a tender point at the usual spot 1 inch above and to the right of the umbilicus, which enabled me to diagnose gall stones in the common duct and to advise operation. Operation, September 21st, 1901. Six large gall stones

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removed from common duct by choledochotomy. Good recovery. A letter in November stated that he had gained 1 stone 11 lbs., that he had completely lost his jaundice, and was quite well.

All these cases go to support the first proposition, that if the diagnosis be doubtful and the patient be at all in a condition to bear operation, exploratory laparotomy should be performed, for not only may a removable cause of the jaundice be discovered, but it may fortunately happen that even if the disease appear to be incurable on exposure, the operation *per se* may have some hitherto unexplained influence leading to recovery, as in two of the cases mentioned and in others that I have seen.

Were it necessary I could very considerably amplify this list of operations in doubtful cases leading to a complete and perfect cure of patients pronounced by experienced and careful observers as hopeless, and in many of the cases the patients had been specially warned by their advisers on no account to consult a surgeon lest he should be tempted to interfere.

The class of cases which I described before the London Polyclinic as chronic pancreatitis demonstrates how unwise it is to give a too gloomy prognosis even in cases apparently malignant, for it is to be borne in mind that the hardness of the head of the pancreas in chronic pancreatitis may resemble in touch that of cancer, and it is only by the subsequent course of events after the operation for jaundice that the true nature of the disease is made manifest by the complete recovery of the patient. Moreover, even in some tumours of the gall bladder inflammatory may closely resemble malignant growth in almost every particular save the one, that after operation the inflammatory swelling tends to disappear and the malignant to increase.

2. *In Malignant Disease.*

The second proposition as to operating in the presence of a positive diagnosis of a malignant disease, so far as such a diagnosis can be positive. There are clearly in my mind two classes of cases in which operation can do no good :—

(1) Primary cancer of the liver, characterised by irregular enlargement of the organ, with the usual general signs of malignant disease ; and

(2) Cancer of the head of the pancreas after middle life, characterised by the entire absence of pain at the onset of jaundice, an

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absence of the history of gall-stone attacks, an extremely rapid loss of weight and strength, and the presence of an enlargement of the gall bladder, and usually some enlargement of the liver.

In the former disease the growth is too diffuse for removal, and in the latter my experience is that the disease is absolutely irremovable, and that drainage of the gall bladder, even if successfully accomplished, does not prolong life materially; and this is borne out by Dr. Hale White's observations published in 'Guy's Hospital Reports,' and in the series of operations collected by Dr. Takayasu, of Osaka. In younger patients the question of operation should, however, always be seriously considered, as malignant disease of the pancreas is not common until middle age or later, and chronic pancreatitis may be the cause of the jaundice, and is curable by drainage of the gall bladder.

The following cases show that in forms of cancer, other than those mentioned, relief, or even apparent cure, may result from operation:—

No. 201.—Woman, aged 52. Pain over gall bladder region for a year; tumour noticed a month. Jaundice. Operation, November 27th, 1897. Cancer of gall bladder and adjoining part of right lobe of liver, which was elongated, removed by elastic ligature. Recovery and out of doors in six weeks. Recurrence of disease following year.

No. 271.—Man, aged 46. Seven years gall-stone attacks. Infective cholangitis. Loss of 4 stone in weight. Jaundice. Operation, June 26th, 1899. Tumour of liver adjoining gall bladder excised by wedge-shaped incision. Fundus of gall bladder removed. Cholelithotrity and cholecystenterostomy. Complete and perfect recovery. Patient in excellent health at present time, two and a half years after operation. Microscope showed disease removed to be cancer.

No. .—Woman, aged 63. History of pain and tumour associated with jaundice. Great loss of flesh and strength. Operation, August 10th, 1900. Mass of growth discovered in liver, gall bladder, and pylorus. Cholecystectomy, pylorectomy, and partial hepatectomy performed. Good recovery. Patient well, and in good health now.

3. *In Gall Stones, &c.*

As regards the third proposition—that if gall stones or any of the first five causes of jaundice enumerated in the opening of my paper be diagnosed, operation is urgently demanded, if the patient be in a fit condition to withstand any operative interference.

I find that I have operated on 212 patients suffering from obstructive jaundice dependent on one or other of the causes

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mentioned. Of these, 183 recovered, showing a mortality of 13·6 per cent.; 60 were the subjects of malignant disease either of the liver, bile ducts, or pancreas, and of these 46 recovered from operation and lived for various periods, some of them being well at the present time, thus showing a mortality of 23·3 per cent. In support of the third proposition, 152 out of the 212 cases were operated on for obstruction dependent on gall stones in the common duct or on other non-malignant causes, with 135 recoveries. Many of these patients were extremely ill at the time, and operation was only undertaken as a *dernier ressort;* but none of these almost moribund cases have been left out of the list, which shows a mortality of 9·8 per cent., an experience which will, I trust, justify my assertion that these cases may be operated on with a very reasonable prospect of success, and that, as a rule, they demand surgical treatment, and at an earlier stage than has hitherto been granted; for it must be borne in mind that nearly every recovery is a life saved, since in the greater number of cases of obstructive jaundice, whether arising from gall stones, hydatid disease, chronic pancreatitis, or stricture or adhesions obliterating the lumen of the bile ducts, death after more or less prolonged suffering is the usual termination.

To give even a brief abstract of every case is quite impracticable in the limited time at my disposal; but many of my cases can be found in my work on 'Diseases of the Gall Bladder and Bile Ducts' (second edition), and others on chronic pancreatitis have been published in the 'British Medical Journal' and the 'Lancet.' I must, therefore, content myself by giving an abstract of a few of my cases, promising that a record of the whole shall ultimately be furnished:—

CASE .—Lady, aged 54; resided in India for 18 years. Characteristic gall-stone seizures with jaundice for two years. Loss of flesh and invalidism. Operation, August 22nd, 1900. Choledochotomy and removal of five stones from common duct. Complete recovery, and patient now well.

CASE .—Lady, aged 57. Several attacks of gall-stone colic in course of years; jaundice a month. Painful and tender tumour. Operation, June 8th, 1901. Choledochotomy, with removal of an oval concretion from common duct, and cholecystectomy for removal of calcified gall bladder containing one stone. Complete recovery. Patient now well.

CASE .—Lady, aged 45. Jaundice and dilated stomach, associated with vomiting and painful indigestion. Operation, May 5th, 1901. Gall stones found in common duct and removed by choledochotomy. Pylorus

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stenosed and treated at same time by pyloroplasty. Complete recovery; loss of all abdominal symptoms. Quite well at present time.

CASE .—Man, aged 60. Great loss of flesh for four years, and painful indigestion. Painless onset of jaundice six months previously. Great loss of flesh. Some enlargement of liver, but none of gall bladder; no nodules to be felt. Exploratory operation advised, but declined. Two years later he saw me again, and was then reduced to an extreme degree of atrophy and feebleness. Liver enlarged, but not nodular. Deep jaundice, varying in degree. Slight ascites. Operation, October 21st, 1901. Cirrhosis of liver, with ascites, and one gall stone found in common duct. Choledochotomy, and at the same time fixation of omentum to abdominal wall. Good recovery; loss of jaundice; gain in weight and strength. Now well.

CASE .—Woman, aged 36. Painful attacks for 15 months, associated with jaundice, which had been continuous for some months. Operation, March 29th, 1900. Cholecystotomy and removal of several concretions from gall bladder and cystic duct. Large stone in common duct crushed between finger and thumb and removed by gall-stone scoop. Complete recovery. Patient now well.

Other cases could be given, of cure by cholecystotomy and manipulating the stones backward into the cystic duct and gall bladder, and thence removing them; of duodeno-choledochotomy, or opening the duct through the duodenum; of adhesions binding down the common duct; of stricture of the common duct treated by cholecyst-enterostomy; of chronic pancreatitis treated by cholecystotomy, and of hydatid disease discharging into the bile ducts and treated by cholecystotomy and hepatotomy, but I will only give examples of three conditions:—

No. 161.—Man, aged 44. Six years' history of swelling over liver, one year's history of attacks resembling gall stones with infective cholangitis and jaundice. Operation, January 1st, 1897. Hydatid disease of liver discharging into bile ducts. Hepatotomy and cholecystotomy. Good recovery and ultimately complete restoration to health.

No. 261.—Woman, aged 42. Jaundice for three months; attacks resembling gall-stone colic for two years; no tumour; ague-like attacks. Operation, May 8th, 1899. Dense adhesions between pylorus; gall bladder and liver broken down; gall bladder shrunken and common duct found strictured by adhesions which were all detached. No gall stones found. Good recovery. Jaundice disappeared before patient returned home. Complete recovery.

No. 286.—Woman, aged 51. Attacks resembling gall stones for three years. Jaundice continuous for 14 weeks. Great loss of flesh. Slight albuminuria. Operation, October 23rd, 1899. Fifteen gall stones removed from gall bladder; large mass, hard and nodular, in head of pancreas. Cholecyst-enterostomy. Complete recovery, and patient now in good health. The tumour of the pancreas was a chronic pancreatitis.

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It would be tedious to give further cases, but these will serve as examples of operation for the various causes of obstructive jaundice not dependent on malignant disease.

A careful study of the causes of mortality in the whole series of cases shows the two greatest dangers to be hæmorrhage and shock, and the two next serious causes exhaustion and sepsis, the accidental causes of death, heart disease, syncope, kidney disease, apoplexy, and other accidents being such as may follow any operation in patients so extremely ill as all cases of obstructive jaundice must be.

The study of the non-successful cases is, perhaps, the most instructive, and from them we can derive lessons that will, I feel sure, tend to diminish the mortality very considerably. This is already occurring materially in my own practice, for the statistics of the cases up to December, 1899, show the mortality as 16·4 per cent., whereas those since January 1st, 1900, though equally serious, have only had a mortality of 14·2 per cent., but in the choledochotomies the difference is as 14·5 to 7·4 per cent. To what is this difference due?

First, I think to increased experience; and, as a consequence, a greater accuracy in diagnosis and a better selection of cases.

Secondly, to knowing when to stop in a case where, after exploration it is manifest that neither cure nor even material relief can result from operation.

Thirdly, to improved technique in operating, and in the preliminary and subsequent treatment of cases operated on; and

Fourthly, to increased practice enabling the operations to be accomplished in little over half the time they once required.

In the heroic employment of calcium chloride we have a very efficient means of modifying the coagulating power of the blood, but to be efficient it must be administered for at least two days before operation, and along with nutrient enemata for three or four days or longer if necessary afterwards. Adhesions should be ligatured where possible, and all bleeding points both in the parietes and inside the abdomen should be clamped and ligatured; or, if that be impracticable owing to general oozing, pressure by means of sterilised gauze affords a useful and safe means of hæmostasis.

In preventing shock, the envelopment of the patient in a suit of cotton wool, operating on a heated table, administering 10 minims of liq. strychninæ *B.P.* before beginning the operation, and giving a

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large normal saline enema, with or without brandy, half an hour before operating and immediately after, and repeating it if necessary, we have very efficient means of treatment. But quite as important as any of these measures, or perhaps even of greater importance, is the cultivation of the habit of operating expeditiously, for it stands to reason that, in any patient so ill as chronic jaundice cases always are, every minute's prolongation of manipulations in the abdomen and of anæsthetisation is adding to the risk, and I know that it is often possible to perform even a choledochotomy in half to three-quarters of an hour. Even in the most complicated cases the operation ought not to occupy much longer than an hour, though I have heard of these operations occupying three or four hours. A simple exploratory operation should occupy only 10 or 15 minutes, and should involve little risk of shock.

Exhaustion and heart failure are best combated by rectal alimentation, by judicious mouth feeding as soon as the anæsthetic sickness has ceased, and by the use of small doses of strychnine administered subcutaneously, when called for by nervous depression or heart failure. A rigid all-round asepsis, the employment of gauze drainage where necessary, and the careful removal of the infected bile as it flows from the ducts, give one security from sepsis.

I have purposely said nothing of the operative technique of choledochotomy, as the subject is already a sufficiently large one for discussion; and I thought more profit would result from a limitation of my paper to the question of surgical treatment of obstructive jaundice as a whole.

Mr. J. H. Morgan said he had not come prepared to offer any remarks, but he had come because he was extremely interested in all the work which Mr. Robson had been doing, and in the papers which he had read before this and other societies. To discuss a paper of such wide extent was no small task. He had been particularly struck by the definiteness of the diagnosis before operation. No doubt that definiteness was largely the result of the author's very extensive experience of these cases, as he evidently had arrived at such a knowledge of their symptoms that he could, with fair presumption, differentiate the ordinary cases of jaundice from the more serious ones, such as malignant disease. He gave encouragement to the surgeon by showing even in the more serious cases one need not despair of doing some good. He wished Mr. Robson could go further and give a demonstration of his operative procedure, because an important part of the paper dealt with the need for rapidity in operating. That he (Mr. Morgan) quite agreed with, for, in all operations on the abdomen, shock, though unavoidable in most cases, could be very much lessened by celerity in operating.

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Mr. Eve said he came as a learner, and as the ground covered by Mr. Robson's paper was so extensive it required a good deal of digesting, and he found that his digestive apparatus had not yet completed its work. He thoroughly agreed with Mr. Robson as to the advisability of operating on all doubtful cases of obstructive jaundice, especially in younger subjects, provided the patients fulfilled the conditions named, *i.e.*, that they were not suffering from serious visceral disease. He thought that was now the general practice of surgeons. Among the great benefits which Mr. Robson had conferred on the profession was one which, until quite recently, was novel, namely, a knowledge of the causes of the chronic enlargement of the pancreas which occurred in certain affections of the stomach, the gall bladder, and the bile duct. Before his paper on that subject was published in the 'British Medical Journal' he (Mr. Eve) had already met with one case in a man who was suffering from chronic ulcer of the stomach with obstruction of the pylorus. At the time of the operation it was thought there was carcinoma of the pancreas. When the patient died—some time afterwards—it was found that there was no cancer of the pancreas, but only chronic cicatricial thickening. With regard to manipulating stones from the common duct into the gall bladder, he had never been able to accomplish that feat. Of course when the stones were at the extremity of the cystic duct, perhaps impinging on the common duct (so as to set up congestion and irritation, and thus producing obstructive jaundice), he had succeeded in dislodging them. In one case he removed a very large number of small stones from a gall bladder, and found at the same time that the common duct and the hepatic duct were full of small stones, but he failed to manipulate them into the cystic duct. The question then arose whether he should proceed at once to open the common duct or should wait, and, if necessary later, perform a choledochotomy. He decided to simply drain the gall bladder, with the result that the stones were discharged through the opening in the gall bladder and *per anum*, and there was complete recovery.

Mr. A. E. Barker said he fancied Mr. Robson had, in preparing his very full *ménu*, had a vigorous Yorkshire digestion in view, but the degenerate Londoners were scarcely up to the gastronomic standard of the North. He congratulated the author on his results, which he regarded as equal to any which had yet been recorded. He (Mr. Barker), too, had been agreeably surprised at the disappearance of masses which he had taken to be cancerous, and this had made him more cautious about giving an unfavourable opinion immediately after operation. Among other similar cases, he remembered years ago when operating on a lady, having come down upon a condition of the gall bladder, which he regarded as carcinoma, whilst close by there was a nodule in a gland. He closed the wound and gave an unfavourable prognosis. The next he heard of the case was a long time afterwards, when her grandchild was brought for operation, and he was surprised to hear that the grandmother was well, and had recovered from her former ailment. He also would have been interested in hearing the technique of the operation, as all surgeons were more or less frequently operating for the condition. Perhaps Mr. Robson would deal with that in another paper. He (Mr. Barker) had long been convinced of the great necessity of keeping such patients thoroughly warm, which he did by means of cotton wool. He had also been careful to make such preparations beforehand as would obviate the least delay during the operation, and he was glad that Mr. Robson had again drawn attention to this point. He agreed also that in recent years surgeons had

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been able to keep up the patient's strength, not only by injections per rectum, but by actual transfusion of salines and carbo-hydrates through the areolar tissue.

Mr. WALTER G. SPENCER said that one of the great difficulties in the subject was the fact that the diagnosis of cancer was made early, and that prevented many cases being seen until they were sometimes too late even for Mr. Robson's skilled hands, who had mentioned that many, not only in general practice, but consultants as well, did make the diagnosis of malignant disease too early. He (Mr. Spencer) was much interested in the statement of the duration of the disease in the cases that had been referred to as having been elsewhere diagnosed as cancer six to nine months previously. On the other hand, the author mentioned a set of cases in which malignant disease came on at a late stage, after a history of gall-stone colic. Malignant disease of the head of the pancreas was so rapid that there could be no difficulty in the diagnosis of those cases; but in a case which had already lasted nine months the presumption was that it was not cancer. The other cases of cancer must be attributed to the irritation caused by the gall stones which had existed for a long time. It was interesting to hear of the discovery of these cases of local cancer and their removal, sometimes with a wedge of liver. Did he understand Mr. Robson to say that in rapid cases of cancer, particularly of the head of the pancreas, cholecystduodenostomy was scarcely worth doing at all as regards the prolongation of life? He (Mr. Spencer) recalled that the earlier statement of Murphy was that six months' prolongation of life might be expected in some cases, unfortunately a too optimistic opinion.

Mr. Robson had said he would hardly operate on cases of ascites, although he had operated on one case and stitched the omentum to the abdominal wall. He (Mr. Spencer) had also seen cases of ascites due to gummata. The President would remember a case of tumour of the liver pressing on the gall duct in which there was some ascites, and in which exploration revealed an unsuspected gumma. Exploration gave one the opportunity of arriving at a diagnosis, and of returning to the medical treatment if necessary. Mr. Robson had not referred to cases where gall stones sometimes passed or ruptured through into the duodenum, Where a gall stone was found in the intestine as a cause of intestinal obstruction, the history was often very uncertain in respect of the jaundice. He had had two cases recently where gall stones had proved to be the cause of the intestinal obstruction, and in which only by putting leading questions to the patient could a history of transient jaundice be elicited. Why did this rupture into the intestine seem to occur in those cases rather early? It did not happen after persistent and long-continued jaundice. At what point did the gall stone ulcerate through into the duodenum?

Mr. HUTCHINSON, jun., said that whilst Mr. Robson and Mr. Barker had urged the extreme importance of rapidity in operations on the abdomen, he ventured to suggest that often rapidity was not of so much importance as other factors, such as careful attention to every detail. For example, in gynæcological operations on the lower part of the abdomen, among the most successful surgeons were those who seemed almost to disregard the item of time, and who would be, from Mr. Robson's point of view, dangerously slow operators. Again, in collecting the records bearing on resection of intestine for gangrenous

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conditions (*see* 'Clin. Soc. Trans. for 1900'), he had not found that shock bore any fixed relation to the time taken in such operations. For instance, the mortality from suture—the slow operation—was much lower than that from Murphy's button. One would have expected that the fatal cases after suture would have died from shock, but it was not so; they lived for three or four or five days; whereas those treated with Murphy's button lived only 24 hours or so. The only fallacy he could suggest was that sometimes the worst cases of all were treated with the Murphy button. Mr. Robson would certainly admit that patients with gangrenous gut were in as grave a condition before operation as those with obstructive jaundice. He was glad to hear Mr. Robson question the utility of operation where gummata were the cause of obstructive jaundice, though such cases formed no doubt a very small minority. It agreed with his belief that, speaking generally, operations on gummatous lesions were best avoided.

Not many years ago it was proposed by Mr. Horsley and others that they should operate on gummata when situated in the meninges and cortex of the brain; and, from time to time, papers appeared advocating operations in the case of gummata in bones and subcutaneous tissues. He (Mr. Hutchinson) doubted whether it was ever worth while to let a patient run any serious risk from an operation when a gumma was the cause of the condition, and especially to excise a gumma from the brain or other important viscus. If a gumma were found, the operation should be stopped, and the patient subsequently treated medicinally.

He would be glad to know what was the utility of stitching the omentum to the abdominal wall as a remedy for ascites. He was himself quite sceptical on the point, and, so far as he could see, the only good it could do was to provide anastomosis between the few small veins of the omentum and those of the abdominal parietes; but one knew how easily such anastomosis was provided by the adhesions which the omentum was only too apt to contract in any abdominal section, unless the wound were sutured in the most careful way. Mr. Robson had mentioned several cases of chronic pancreatitis which were improved by cholecystotomy, but he could not wish it to be understood that all such cases were benefited by the operation. Chronic pancreatitis sometimes had nothing whatever to do with the condition of the gall bladder or biliary ducts. In one very marked case, where the pancreas was as hard as a stone and lumpy, feeling exactly like scirrhus, he operated and found the condition was due to old extensive ulceration of the anterior and posterior walls of the stomach, gluing the stomach to the liver, and obliterating the lesser cavity of the peritoneum. There could have been no possible use here in performing cholecystotomy. Where there was no distension of the gall bladder, he took it that Mr. Robson would not think it worth while to perform cholecystotomy in chronic pancreatitis. He was specially interested in the narration of cases of inflammatory trouble simulating cancer. Although the result of exploratory operation in such cases might appear to be brilliant, it must be remembered that such patients would often do just as well without it. For example, three years ago he saw a gentleman who had ascites and extreme loss of flesh with jaundice. Large lumps could be felt just under the ribs, evidently connected with the liver. Malignant disease was diagnosed, an exploratory operation was discussed but not performed, and he, in common with other surgeons and physicians, gave a gloomy prognosis. Yet the patient got practically well. There was no evidence of syphilis in his case, and it was an unusual example of hypertrophic cirrhosis. His recovery was attributed to a stay at one of

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the baths, and a long course of taraxacum. To do an exploratory operation in every case of malignant disease, especially in private practice, would justly lay one open to criticism. He highly appreciated Mr. Robson's admirable paper; its author spoke as one of unsurpassed experience, either in this country or abroad, on the subject under discussion.

Dr. VOELCKER said that though the chief interest of the paper was for the surgeon, it was one which physicians might listen to with great benefit, whether because of diagnosis, or on account of the brilliant results which had been obtained, or the lessons which one learned as to the use of measures other than those which were purely operative in securing the ultimate success of operative procedures. It was consoling to find that in the difficult cases to diagnose Mr. Robson had shown that it was possible to arrive at an opinion. Many a physician felt that at the present day there was a danger in not attempting to make a diagnosis because a surgeon was called in to make a diagnosis for him. That was to be deplored, especially as Mr. Robson had shown how successfully a diagnosis could be made before operating. Physicians might learn a great deal from surgeons in the matter of drugs and their administration. Sometimes a heroism was exhibited by surgeons in that respect which perhaps physicians did not often show. He would be glad to hear from Mr. Robson in what doses he administered chloride of calcium with the view of diminishing the risk of hæmorrhage after the operation. While he (Dr. Voelcker) was house surgeon under Mr. Barker, it was noticed how greatly prevention of exposure lessened shock following operation. That was specially so in the case of children.

Mr. MAYO ROBSON, in reply, said he had not time to deal adequately with the various points raised. For a long time he did not give chloride of calcium in larger doses than 15 grains; but he then got to 30 grains, which he thought was all that was required, if given three times a day, for two days before the operation. Immediately after the operation he usually prescribed a pint of saline solution into the bowel, into which solution was put a drachm of chloride of calcium, and a drachm was given per rectum three times a day for two or three days subsequent to the operation. On one patient he did an operation for chronic jaundice, and removed several gall stones from the common duct. He had given chloride of calcium two days before, and there was no oozing of blood at all, and only the pumping vessels had to be tied. The nurse had omitted to inject the chloride of calcium afterwards, and the second night his house surgeon was called up and found the dressings deluged in blood. He opened the wound, and found the stitch punctures were all bleeding. He immediately gave, on his own responsibility, a drachm of chloride of calcium, and repeated a drachm every two hours for several doses, and then ordered it in 30-grain doses every four hours. That completely stopped the hæmorrhage. Since then he had seen it given in drachm doses without any harm. Professor Wright, of Netley, introduced it, and used it on dogs, and he (Mr. Robson) thought he was the first to use it in surgery. Wright pointed out that after saturating a dog with chloride of calcium he could divide the jugular vein and the blood would coagulate as it flowed out. He (Mr. Robson) used it because he had been troubled by one or two cases which died from the oozing of blood after operation.

In reply to Mr. Eve, it was very rarely that gall stones could be manipulated back from the common duct; but sometimes the gall bladder,

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cystic duct, and common duct were crowded with gall stones, and then it could be done. In a recent case he removed 88 gall stones from the common duct, and several of them he manipulated through the dilated cystic duct; but there were so many that he removed the others by choledochotomy.

In reply to Mr. Barker, they were all puzzled by those cases which were cured by operation *per se*. Mr. Hutchinson thought they might get well without operation, but of that there was doubt. Three days ago he had seen a patient for whom he had done a colotomy and exposed the sigmoid flexure. He found a nodular tumour in the upper end of the rectum and in the lower sigmoid. There was now no tumour, and the patient said he believed there never had been a cancer. He hoped to explain the technique of the operation of choledochotomy in a future paper.

With regard to intestinal obstruction from gall stones, he believed that in most of those cases it was due to large stones ulcerating from the gall bladder or cystic duct into the duodenum. The jaundice which occurred was catarrhal or from pressure, and not from the obstruction of the common duct by the gall stone. Frequently in those cases there was no history of gall stones, because there had been no obstruction to the flow of bile, and no impaction of concretions. Those large gall stones never got impacted in the cystic duct, and did not produce pain, unless it were that of chronic catarrh.

With regard to ascites, if that were present and were due to syphilis, or to tubercle, or even cirrhosis, the patient might get well of it. If he found it dependent upon malignant disease, he would close the abdomen right away, and he did not think stitching the peritoneum to the surface of the abdomen would do any good. He had stitched up the peritoneum in several cases of ascites with benefit, but it was early to decide whether that procedure would stand the test of time.

In regard to cancer of the head of the pancreas, he was clear, from an experience of 25 cases, that operation did no good, and if he arrived at that diagnosis he did not operate. The prolongation of life in the recorded cases was very little; moreover, those patients stood operation very badly. Out of 30 cases of chronic pancreatitis he had had only one death, whereas in 25 cases of cancer of the head of the pancreas he had lost nearly half directly from the operation, and those who recovered did not have any great prolongation of life.

He differed from Mr. Hutchinson with regard to the importance of rapidity in operating. He did not mean undue haste, but care with expedition. Rapidity of operating was of much less importance in the lower abdomen, as was shown by Crile's book, based upon operations upon animals. Taking a large number of the cases, he felt sure that the more quickly they could operate the more safely they would do so. Time was a very important item in obviating shock.

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January 27th, 1902.

ON OBESITY.

By Sir LAUDER BRUNTON, M.D., F.R.S.

THE subject for discussion to-night is that of obesity, but I feel that first of all I owe an apology to the Society for attempting to introduce it when we have in our President, Dr. Allchin, one who has studied it much more thoroughly than I have done, and who could have introduced it so much better than I. My apology to you is that his position as President prevented him from opening the discussion, and it is by his request that I venture to do it.

We may begin the discussion by asking what is obesity? It is an excessive amount of fat, causing the patient to be too large to the eye, too heavy to the scales, or leading to discomfort and distress. It is sometimes difficult to give an exact definition of obesity. When a man is so stout that his body and limbs have become almost shapeless, there is no difficulty in recognising his case as one of obesity, but there are individuals who, though considerably stouter than their neighbours, are yet brisk, active, and capable of much exertion both of body and mind, the unusual amount of fat which they possess seeming to be no hindrance to their activity. One can hardly call those persons obese, and yet these very people after a short but sharp attack of influenza, which weakens the heart, may feel their weight cumbersome, and if we saw them for the first time in such a condition we should call them obese without hesitation. One may say in general terms that obesity commences when the patient's fat is obviously very much greater than normal to the eye, or when it becomes an inconvenience to himself. In order to define more accurately what I mean, it may perhaps be well to give a few numbers. The average height of an Englishman of 25 is said to be five foot eight (68 inches), and his weight ten stone five (145 lbs.). These numbers remain pretty nearly constant up to the age of 40, when the weight generally begins slightly to increase. Germans of the same age are on an average about one and a half inches taller, and weigh 5 lbs. more, while the Latin races are about 2 inches less and 7 lbs. lighter. According to Van Noorden this weight

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may increase as much as 15 per cent. and still be within the normal limits. When it reaches nearly 30 per cent. one may regard them as stout, from 30 to 35 per cent. too stout, from 35 to 50 per cent. as moderately obese, and above 50 per cent. as very obese. The accumulation of fat occurs specially in the subcutaneous tissue, in the omentum, and around the heart and kidneys. Accumulation of fat in an animal or a man may depend on one or two causes: first, on too much fat or fat-forming food being taken into the body; second, on too little being burned off. These two causes may, of course, act together, and very often do. Fat consists chiefly of mixtures in different proportions of glycerine united to fatty acids. These mixtures vary in different animals, but each animal keeps its own fat pretty nearly up to a constant standard, although it may feed upon other animals having a different kind of fat. This is not altogether the case, however, for, as an old nurse of Weir Mitchell remarked, "some fats are fast, some fats are fleeting, but cod liver oil fat is soon wasted." Her keen observations showed that all fats are not alike, and this has been demonstrated by various scientists who have fed animals on fat which could easily be recognised, and more especially on a kind of fat from rape-seed oil, which has an entirely different composition from most other fats, inasmuch as glycerine is absent from its composition. Animals have not only the power of forming their own peculiar fat when they are fed upon foods of a different fat, but they are able to form fat from different kinds of food, even if fat itself is not contained in them. They can do this from foods in which fat is not contained, such as albuminates and carbo-hydrates. Under albuminates are included the flesh, that is, the muscles voluntary and involuntary of molluscs, fish, fowls, and animals of all sorts—as well as eggs—and, although not properly albuminous, tendinous structures, such as gelatine, may be included in this class. The class of carbo-hydrates includes all kinds of sugars and farinaceous stuffs, and plants containing them, as well as cellulose, or, as it is sometimes popularly called, woody fibre. But a distinction must be formed between cellulose and starches or sugars, though it consists like them of carbon, associated with hydrogen and oxygen in such proportions as will form water, and is thus a carbo-hydrate. For although sawdust may be converted by chemical means into sugar, yet, physiologically, sawdust and other forms of cellulose are not capable of being digested to any

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great extent in the human organism, and therefore cellulose is not a food like starches and sugars. As the vital functions in human beings are maintained by a process of combustion, the nutritive value of foods is reckoned to some extent by the heat which each kind will give on combustion. The most convenient unit is the calorie, or amount of heat required to raise a kilogramme of water through one degree centigrade. The heat value of a gramme of dry albuminose is 4·1, that of carbo-hydrates is the same, whilst that of fat is no less than 9·3. But the body not only requires food which will yield energy, it requires also material to supply waste, and whilst albuminous materials will do both, carbo-hydrates and fat do not supply material for repair. It is therefore possible for a man to live entirely upon meat and water, but he cannot live entirely upon sugar and fat. Thus, although the nutritive value of albumens and of carbo-hydrates is the same if estimated by the quantity of heat they yield in combustion, the part they play in nutrition is entirely different. The body splits off fat with difficulty from albuminous materials, such as lean meat, but it forms fat with considerable ease from sugar and starch. The tendency to lay on fat is therefore determined, to a considerable extent, by the *nature* of the food. Individuals or races which live upon much animal food tend to be thin and wiry, while those who live much upon starchy foods and sugars have a greater tendency to plumpness. There is also a considerable difference in individuals, for just as some grates will heat a room with much less coal than others, so some persons appear to get through a good deal of work, both mental and bodily, on a small amount of food, and always look well nourished, while others who apparently work less and think less, yet eat enormously, and remain thin and hungry looking. Such individual differences probably depend, to a considerable extent, on the power of the tissues to use up the nutritive materials absorbed into the blood, and such power probably depends to a great extent on the presence of a larger or smaller quantity of enzymes. The tendency to an accumulation of fat, both in men and animals, after the removal of the testis or ovaries is well known, and in some experiments on the subjects Loewy and Richter found that in dogs the administration of ovarian extract had no effect when the generative glands were present, but increased oxidation in animals from which the ovaries or testicles had been removed. In these experiments they did not

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find testicular extract have the same action. But such individual peculiarities, although of occasional importance, have much less to do with obesity in general than excessive food and deficient exercise, or a combination of both. The great majority of people, in this country at least, begin to get stouter in middle age. About this time also they become less active. While taking less exercise, they not infrequently go on taking as much food as they did when younger, or even more, because they either have attained a position where they can afford to live better, or they indulge in the pleasures of the table in place of the active amusements which they previously had enjoyed. Under these circumstances they tend to become much fatter and heavier than before, yet while bodily exercise is more fatiguing and less enjoyable, their intellects may remain quite as bright as before, and if engaged in occupation where mental activity is necessary they may be able to do their work as well as ever they did. It is usual to distinguish two forms of obesity—the plethoric and the anæmic. A most striking example of the former kind was familiar to the members of this Society some years ago in the lamented Dr. Milner Fothergill. Although so stout that on one occasion he stuck in the door of a railway carriage, and could neither get in nor out, his mental activity was very great, and his muscular strength was also very great, whilst the ruddy colour of his face and mucous membranes showed that his blood was rich and his circulation perfect. He told me that on one occasion he had made an attempt to reduce his weight. This he effected most successfully, so much so that he was not only able to run, but he could vault a five-barred gate with ease. He found, however, that when his bodily activity increased his mental activity diminished, and he deliberately gave up the attempt to keep himself in training, because he found that if he did so he would have to sacrifice all hope of attaining eminence in his profession, and be content with the life of an ordinary practitioner. The other type of obesity, the anæmic, is more often found in women whose colour is pale or tallowy, whose mucous membranes are pale, and whose muscular strength is small, and who are usually neither active in body nor mind. Such a condition sometimes comes on after profuse hæmorrhage, and may be due in considerable measure to the loss of blood. In some parts of Germany it is the custom to bleed cows in order to fatten them, and, according to the researches of Bauer, the abstraction of

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blood from animals seemed to lessen the tissue change, and he explained the accumulation of fat after bleeding on the hypothesis that less oxygen was carried to the tissues, and so the fat underwent slower combustion, and consequently tended to accumulate. Some doubt has been thrown on these researches by later observers, and it has been suggested by Van Noorden that the fattening after loss of blood, and in anæmic conditions generally, is not due specially to deficiency in oxidation, but to the lessened muscular exercise which men and animals take in such a condition on account of the discomfort which they feel from the shortness of breath and languor on any exertion. It is possible that there may be a certain amount of truth in Bauer's theory, but there can be very little doubt that anything that interferes with the action of the heart tends to increase the accumulation of fat by lessening muscular exercise. Febrile disease, such as typhoid, may cause much emaciation of the patients during its progress, but after the disease is over the patients may become considerably stouter than before, partly from the increased food forced upon them to restore their strength, and partly because they eat more food to restore their strength, and they take less exercise because they are still feeble. Other diseases which lessen activity tend to cause accumulation of fat, such as chronic nephritis, on account of the languor it produces; chronic bronchitis, and emphysema, with shortness of breath on exercise; gout, rheumatism, or injury to the legs, on account of the pain and discomfort in moving, all tend to lessen the patient's activity, and consequently make him stouter. And then he enters a vicious circle. With increasing weight there comes increasing difficulty in moving. The enfeebled heart, the labouring lungs, the rheumatic muscles, have all a greater strain to bear, and the tender joints have a greater weight to sustain. Nor is the fat usually distributed evenly over the surface of the body. It tends to accumulate in those parts where the adipose tissue is normally thickest, but it has a special tendency to accumulate in the cavities of the body, in the omentum, round the kidneys, and round the heart. The breathing space is thus still further lessened, the movements of the diaphragm are restrained, the heart, encumbered by fat, acts with more difficulty, whilst the bowel, laden with fat and compressed with less energy by the thicker and feebler abdominal walls, acts with less vigour. The patient's circulation thus becomes feebler, his breath shorter, and

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his bowels constipated. Moreover, the heart and liver become not merely imbedded in fat, but more or less infiltrated with fat, and the cardiac muscular fibres may even undergo fatty degeneration. In this way the general circulation becomes still feebler, and the circulation through the liver being impeded by fatty enlargement of the liver cells, the patient becomes liable to attacks of hæmorrhoids, a condition still further increased by the constipation from which the patient so frequently suffers. The impeded respiration, and the efforts required to make these slight movements, lead to much sweating, whilst eczema, boils, and carbuncles still further increase the discomfort of the patient. The trouble and discomfort which the patients experience almost make their lives a burden to them, and, in addition to their other troubles, they suffer from depression of spirits.

As the cause of obesity, either in this severe or in slighter forms, consists in the ingestion of more fat-making food than the body can burn off, its treatment naturally consists in limiting the food both in quantity and quality, and increasing the combustion of the body by accelerating the circulation. The best way of stimulating combustion is undoubtedly muscular exercise, but in severe cases we are met with the difficulty that the patient is unable to take muscular exercise. Here comes in the use of massage, by means of which the circulation through the muscles, as Tunnicliffe and I have shown, may be increased three-fold, and thus the effect on tissue change obtained without any inconvenience to the patient. Moreover, by massage the work of the heart is considerably lightened. Usually the heart has to complete the circulation, and to drive blood from the left ventricle back to the right auricle; but massage takes over part of the work from the heart, and by undertaking the venous circulation for the time being, and passing much of the venous blood on from the extremities to the centre. At the same time, the skilful application of massage to the abdomen will lessen or remove constipation, which may be present. But massage alone is not sufficient. It ought only to be the preliminary to more active exercise. Until the heart is stronger the exercises should be those of gradually increased resistance, such as have been brought into vogue by Schott, at Nauheim, and as the strength increases the patient should first walk along the level ground for greater and greater distances, then up steeper and steeper inclines, according to his ability. Such walks have been laid out in this

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country at Llangammarch, in Wales, and one of the best places that I have come across for them abroad is Badenweiler. But the great difficulty that one meets with in London, especially with the slighter cases of obesity, is that of time, because many men who are gradually becoming stouter cannot afford the time for walks. Some of these I find get great benefit from an hour in an institute presided over by someone who understands the treatment and can regulate it according to the requirements of each patient, and a good many City men find that it is worth their while to take an hour every day in an institute of this sort on their way to the City. But even an hour is more than some can give up, and for them I think the best thing is exercise with one of Sandow's or Whiteley's exercisers. When they are getting into good condition a punching ball becomes very useful, as they are able to take a great deal of exercise in a very short while with it. They can do this just after rising, and in 10 minutes or a quarter of an hour they will probably be perspiring, and they can then take their bath and dress. A Turkish bath once or twice a week is often both a luxury and a benefit. But exercise alone rarely suffices to bring down obesity, because exercise in itself tends to increase the appetite, and in persons who tend to get too stout, appetite not only follows but overruns the effect of exercise, and instead of getting thinner they become stouter. Excepting in institutions it is very difficult, and sometimes almost impossible, to have the solids and liquids taken by a patient weighed and measured, but the amount of food can generally be lessened very greatly by simply insisting that solids and liquids shall be taken at different times. Many people are apt to wash down food, and especially farinaceous food, without much mastication, but if the farinaceous food is of a dry kind, such as crisp toast, rusk, or biscuit, the quantity that can be got down in a given time, if fluid be withheld, becomes very much less. I am inclined to fancy, but perhaps on insufficient grounds, that there is some difference in digestion in the stomach and less tendency to form fat than when the digestive process is carried on in the intestine. The diet that I am accustomed to recommend to my patients who tend to get stout is the following:—

On awaking in the morning, sip a tumblerful of hot water with a slice of lemon in it if desired, and if the bowels are constipated half a teaspoonful or a teaspoonful of *pulverförmig* Carlsbad salts may be added to the hot water.

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For breakfast take lean meat, either hot or cold, steak, chop, or cutlet, potted meats, kidney, tongue, chicken, or game, hot or cold, boiled, roast, grilled, or devilled. Fish of any kind, except herring and mackerel. The lean of bacon or ham, hot or cold. Eggs may be taken boiled, poached, or as omelet, but rather sparingly. Towards the end of the meal a breakfast cup of tea or coffee, sweetened with saccharine, may be taken. It is better without milk or cream, but still a very little may be allowed of either unless the tendency to stoutness is exceedingly great. During the meal about 1 ounce of dry toast—that is, one slice about 5 inches long, 3 broad, and ¼ inch thick—may be slowly eaten and thoroughly chewed. If more bread is wanted, almond or gluten biscuits, gluten bread, or gluten rusks should be taken.

Between 11 and 12 a tumbler of hot water should be sipped, as already directed.

For lunch, fish, fowl, or game of any kind may be taken as at breakfast. One helping of vegetables of any kind, excepting potatoes. A baked or stewed apple may be taken, sweetened with saccharine, and a glass of claret, chablis, or hock. If the wine gives rise to indigestion it should be omitted, or a tablespoonful of whisky or brandy in a half to a whole claret-glassful of water may be substituted. The whisky or brandy should be carefully measured in a medicine glass and not in a spoon, for otherwise there may be a tendency to involuntarily increase the quantity. Gluten rusk, gluten bread, or gluten or almond biscuits may be taken if desired.

Between 4 and 5 hot water may be taken with tea, as already described, and an almond or gluten biscuit or gluten rusk may be taken with it.

At dinner fish, flesh, or fowl, vegetables, and apple may be taken as at lunch under the same restrictions. Just after dinner a cup of black coffee, sweetened with saccharine, may be taken.

At night a tumbler of water, to be sipped as already described.

In this dietary the quantity both of food and liquid is practically left at the patient's discretion or appetite, but the quality is much the same as that of a diabetic dietary, with the fat omitted. Should such a dietary as this, combined with a fair amount of exercise, not succeed in reducing the patient, it is, I think, best either to send him into a nursing home, where the quantity can be carefully regulated as well as the quality, and where exercise can be systematically carried

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out during a much longer period of the day than when the patient is at his or her ordinary avocations. But if this is not practical, then I think the next best thing is to put the patient on the so-called Salisbury treatment, which is very much like what was formerly recommended by Harvey and Banting, plus the addition of hot water. The Banting treatment practically consists in starving the body of fat-making material by feeding entirely on meat, from which fat is formed with difficulty. The disadvantage of this treatment consists in the fact that while little material is afforded for the formation of fat, an excessive amount of nitrogenous waste products occurs in the blood, and unless they are freely eliminated may produce poisonous results, such as languor, nervousness, and insomnia. By taking a large quantity of hot water between meals the excretion of the products of muscular waste is greatly quickened, and this danger is to a great extent averted. The Salisbury system, as expounded by Mrs. Stuart, is to take lean beef and minced beef only for meals, and take hot water between times. The meat should be freshly killed and finely minced by sending it through a sausage machine three times; all the fat, gristle, and connective tissue should be removed; the minced meat made up lightly into cakes, seasoned with pepper and salt, and moistened with good soup and grilled. Or the minced meat thus prepared may be mixed with cold meat soup, free from fat, in the proportion of 1 pint to 1½ lb. of mince, and heated over a gentle fire till thoroughly cooked, but not brought to the boil. If the patient cannot take mince-meat alone, some small digestive biscuits, or bread roasted crisp in an oven, and butter may be taken in the proportion of one mouthful of either of these to four or six of the mince. Such a dietary as this may sometimes be carried on for two or three years without any bad result, but where the patient, not knowing the reason for the hot water, has limited this and still continued the mince-meat, disadvantageous results have arisen, just as in the Banting system. There is no doubt, I think, that the use of thyroid extract increases tissue change, and will reduce fat considerably, but I think in most cases it is advisable to diminish obesity rather by attention to feeding and exercise than by the use of thyroid. I have seen the nervousness, tachycardia, and tremor, so characteristic of Graves' disease, produced by patients taking thyroid too long without supervision.

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Dr. BURNEY YEO thought there was very little in the admirable address to which they had just listened that anyone would take exception to, and he could not hope to add much to what Sir Lauder had said. He (Dr. Yeo) was inclined to divide cases of obesity into two classes—the one easy to treat, and the other difficult. One class he would speak of as obesity from "a vice of metabolism," which would include the anæmic cases and those which were hereditary. It had been estimated that 50 per cent. of cases of obesity had been traced to heredity. In the other class one had to do with cases which arose from dietetic errors, and such persons usually became obese at about middle age. A third class could be made, in which the two causes he had mentioned could be combined. He thought there was an analogy between those cases of obesity which were difficult to cure and grave cases of diabetes, and, on the other hand, between obesity easy to cure and cases of glycosuria. By taking away the carbo-hydrates from either the dietetically obese or the glycosuric, the respective conditions were got rid of. But that was not the case where there was defective metabolism. In regard to the cases of obesity which could be easily cured it was most important, if success was to be achieved, to begin early. When a man of 40, who was usually 11 stone in weight, began to find his waist buttons bursting, and the scale showing him to be 12 or 13 stone, that was the time to begin treatment. Some patients would respond in the most precise manner to little corrections in diet at that stage, such as leaving off tea and coffee, and eating fewer potatoes and bread.

With regard to anæmic obesity, frequently met with in young women, in many such cases there was a certain want of ingenuousness. They would declare that they ate practically nothing. He had such a case under his care a few years ago, and got a medical man at one of the German spas to take her into his house as he wished her habits to be noted. A clergyman and his wife were visiting at a town a few miles away from the spa, and the young lady begged to be allowed to go and spend a night with them. But in the night she got up, made her way to the railway station, and booked to England. She did not go home, but went to the house of an indulgent old nurse, where she was. no doubt allowed to follow whatever desires she had. But after she left the spa the medical man received an enormous bill which she had run up with the confectioner.

One point in the treatment to which Sir Lauder Brunton had not alluded was that of an absolute milk diet. Professor Despres, of Paris, had published several cases in which he had produced very considerable loss of weight in obese people who were troublesome to treat otherwise. In one of the cases the weight was reduced by 54 lbs., the patient being kept in the hospital for four months, and limited to a daily allowance of one quart of milk. He (Dr. Yeo) had a patient who was not very obese, and who came with symptoms of gastric ulcer. He kept him in bed a fortnight, and gave a milk diet. In the first 10 days he took 36 ounces of milk per 24 hours, practically nothing else. At the end of a fortnight he had lost 2 stone. He also said he felt very much better, lighter, and stronger. That seemed to be a very easy way of treating obesity. But if that treatment were adopted it was most necessary that the patient should be kept in bed, or quite at rest; the muscles must not be called upon to do any work.

A very interesting question with regard to obesity was its relation to the consumption of water. One of the oldest methods of treating the

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obese was by limiting the quantity of water; but that was not always safe, because many obese people were gouty, and required to take large quantities of a solvent like water for eliminative purposes. He agreed with all Sir Lauder had said with regard to the modes of introducing water for daily consumption in cases of that kind. No doubt people who did not drink with their food ate less, and the masticatory energy expended was greater. It had occurred to him that civilised man was the only animal that drank when eating.

Sir Lauder had spoken of the influence of castration and of the ovarian fluid in relation to obesity. He knew it had been suggested that castration probably led to the removal of the internal secretion which regulated the metabolism of fatty substances. But he thought there was another view—namely, that after castration there was a loss of the sexual emotions, and nothing so tended to make people thin as the worry and anxiety of emotion. It was also well known that obese persons were noted for their calmness; they were not, as a rule, passionate people.

With regard to the use of thyroid extract, he thought that if used at all it should be limited to persons who could not be made thin by dieting, those in whom it might be assumed there was some "vice of metabolism." He would not himself care to give thyroid extract to people who were fat simply through errors of diet, so that they might continue in those errors. If people would not walk in the ways of physiological righteousness, he had no inclination to help them by the administration of thyroid extract, but would reserve that for those anæmic cases which did not respond to dietetic measures.

He was glad Sir Lauder was one of those physiologists who admitted that fat did fatten; some writers on the subject seemed to throw doubt on that point. They devoted all their advice to getting rid of the carbo-hydrates, neglecting the fats entirely.

He would have liked to have heard the opener's experience on the influence of alcohol in the production of obesity. His own opinion was that alcohol was a very important agent in the maintenance of obesity, and that very often dietetic measures failed because there was not a rigid exclusion of alcohol from the daily ingesta. He thought the Salisbury treatment had many advantages, but it was a risky treatment to apply to old people, in some of whom it caused cardiac weakness, which was sometimes alarming. The Salisbury treatment, like every other, had its strict limitations.

Professor SCHOTT (of Nauheim) said the subject was such a large one that it would require a long time to deal with it fully. Therefore he would speak chiefly about fatty heart, and say a few general words in conclusion. There were three different forms of fatty heart—namely, fat surrounding the heart, fat between the muscular tissues, and fatty degeneration. Sir Lauder Brunton had pointed out how difficult it was to say when obesity was beginning. In Germany, 10 or 15 years ago, many thought that when the circumference of the abdomen was greater than that of the thorax the person was obese. But that rule no longer holds good. In many of such cases he found there was nothing but a certain amount of flatulence around the colon and stomach, and when that was relieved there was quite a different measurement. Obesity might be treated in two ways: first, by means of medicines. Not long ago purgatives in large quantities were the rule, and reduction in weight was quickly attained. Then there was the treatment by iodines. In young obese patients with strong hearts and muscles one could risk something,

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but, nevertheless, treatment by iodines was not without risk. He referred to the cases of two patients who had lost weight too quickly, and to too great an extent. They were both tall and strong men, who came to him about 18 years ago. They both had undergone a Marienbad cure, and lost there in the course of four or six weeks 25 and 30 German pounds respectively. Returning home, they continued to lose tissue rapidly, one lost 54 the other 45 German pounds in three months. The result was that when they arrived at Nauheim he found they had fatty degeneration of the heart. This was owing not only to the loss of fat in the abdomen and in the thorax, but also to the loss of muscular and nervous tissue and blood. In other cases obesity was treated by thyroid and ovarian extract. When patients lose too much weight in a short time they are liable to fits of weakness, to dilation, or even degeneration of the heart; and he had seen cases in which the nervous system was so much affected that patients had to be placed in a lunatic asylum. If it be necessary to bring about reduction of fat, this had better be effected by means of diet, exercises, and baths. As to the latter, he preferred cool, carbonic-acid baths to Turkish baths, for after the latter he had seen some bad collapses, especially where the heart was weak. The exercises prescribed by his brother and himself were so well known that it was not necessary to enter into details. Then there was the climbing cure, which had been introduced by the celebrated Dublin Professor Stokes as far back as 1852. Later Oertel had combined this climbing cure with restriction of liquids. As to this restriction, Schott and many others had made experiments which proved that people accustomed to take liquids with their meals eat less as soon as liquids are withdrawn, and of course the final result must be a reduction of weight. There had been different diet cures, but he would only mention two. One was Ebstein's, who gave fat. Dr. Yeo mentioned a purely milk diet, but if the milk was fat enough and the quantity very large, there could not be much reduction. The other was the Schroth cure, giving only dry rolls with red claret. Then Schroth took away the claret, and allowed the patient as many rolls as he could eat, with the result that his appetite failed. It was only a hunger cure, and the milk diet was a half hunger cure. He came to the conclusion that young people might be subjected to this radical cure without any danger, but patients in an advanced age or suffering from any abnormality or weakness, must have their obesity reduced cautiously, and only to a moderate extent.

Dr. Robert Hutchison thought the most interesting question raised that evening related to the cause of obesity rather than to its cure. It was very well to say, as Sir Lauder Brunton had truly said, that obesity was due to one or two causes, or to a combination of them, viz., eating too much, or working too little. But one had to face the question, was not there a group of patients who were more economical machines than other people? Both the opener and Dr. Yeo were evidently advocates of that view. Sir Lauder attributed the low consumption of food for the same output of energy to the rather ubiquitous enzyme; while Dr. Yeo, with more caution, attributed it to a vice in the metabolism. He (Dr. Hutchison) did not think that either of those explanations was satisfactory, because the matter had been put to the test of fairly exact experiment. There was only one of two ways conceivable in which one person might be a more economical machine than another. One was that he should be able to do a certain amount more of work with his muscles for the same consumption of food. But the law of the conservation of energy

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made that out of the question. Therefore one had to fall back upon the explanation that persons who were more economical must produce less heat than others did. That question could be determined, and in Germany patients had been submitted to experiment, and their heat output ascertained. But he did not think any experimenter had found that those persons did put out less heat than others. Quite recently a Dr. Jaquet was said to have found that such people absorbed food more easily than others. He found that the stomach and intestines seemed to perform their work more economically than in ordinary healthy individuals, the saving being 10 per cent. to 25 per cent. But the limits of error in calorific experiments were great, and a small gain, within the limits of error, if spread over a long time, would gradually lead to an accumulation of fat. If such economy in metabolism did take place, obviously the natural cure was not to restrict the intake of food nor increase the output of energy; but one would like some drug which would increase the oxidation of the body, and people thought such a drug had been found in thyroid extract. But it was found that thyroid did not only increase the oxidation of fat, but at the same time the oxidation of albuminous materials; therefore people to whom thyroid was administered suffered from weak heart and loss of muscle, and from the circulation being flooded by excessive nitrogenous waste, and that threw a strain on the kidneys. In practice it was wise to investigate the patient's excretory organs before starting to give thyroid at all, particularly if he were stout.

The influence of fluids on obesity had been mentioned by all the previous speakers. So far as he knew, experiment had not shown that the mere restriction of fluid in itself tended to produce a loss of weight. The restriction of fluid was a method first introduced by Dr. Dancel in France, and afterwards popularised in Germany. It probably owed its success in Germany to the fact that there the restriction of fluid was synonymous with the restriction of beer. That did not apply so much in this country.

Mr. C. B. Keetley said that, as a surgeon, he had for many years taken great interest in obesity. When, 23 years ago, he was appointed surgeon to the Surgical Aid Society, he came into contact with many people who suffered from irreducible herniæ. He read Mr. Banting's own little pamphlet about himself, in which he remarked casually that an umbilical hernia from which he suffered disappeared under his treatment. He (Mr. Keetley) made use of that hint extensively, and in some cases he saw great benefit derived from putting people with severe irreducible umbilical herniæ on the Banting system. He also found it a good preparation for operation for radical cure. A few years afterwards he found that Mr. Bransby Cooper treated cases in the same way half a century ago. Mr. Banting said he got his idea from Mr. Hinton, of Guy's Hospital. He thought it would appear that Mr. Hinton obtained it from Mr. Cooper. In general surgery obesity often proved a serious hindrance, not only in operating, but in diagnosis. He was certain there were many who died of carcinoma of the large intestine who were really sacrificed because their stoutness did not allow of the detection of the tumour early enough for operation. Dr. Mouillot had just suggested to him that some of the fat of such cases should be removed by surgical means. There was something to be said in favour of this, and especially in people of a certain age, as the skin in them became almost as superfluous as the fat. Operation could only be applied to localised collections of fat; but women sometimes felt an inconvenience, and even shame, from

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the amount of fat on the abdomen. He could call to mind one lady who had huge folds of fat hanging down from the abdomen and from each limb. If her lungs and kidneys were found to be sound, there seemed no reason why she should not be relieved of some of them. He suggested that it was not necessary, when laying down tables of diet, to go too much into the nature of the solid food. Frequently experiments were made accidentally. For instance, one had watched such cases as those of corrosive acid poisoning, in which the patients could not take as much food as was good for them, and they emaciated with very great rapidity, in spite of the fact that the food which they took and assimilated most easily was farinaceous food, mixed with milk and water. One reason for not particularising the solids was that it was a trying ordeal for a fat person to diet herself according to a very narrow prescription, and the more freedom they could be allowed, the more likely they were to go through with it. The same remark applied to fluids. In his excellent book Dr. Yeo, for example, had suggested that a patient should take a little hock, a little Moselle, or a light claret; but people who did not pay income tax, and they were the majority, could not be expected to drink wine. He believed the growers of wine on the Rhine would be quite shocked at mixing things so different, in their eyes, as hock and Moselle. One grower of Rhenish wine said he did not know the taste of water, and when asked by a friend what he cleaned his teeth with, he replied contemptuously, "With a little light Moselle." There were other people besides this gentleman who did not know the taste of water. All he could say was that the sooner they acquired that taste the better for themselves. Would it not be better to give a person who was dieted for obesity a free hand to drink good water, and say nothing about claret, or Moselle, or hock?

Dr. Luff said the Fellows were indebted to Sir Lauder Brunton for his extremely commonsense introduction of the subject. If there was any morbid condition the treatment of which should fall within the province of the usual medical attendant, it was the treatment of obesity; it was absurd to make it a speciality, requiring, as it did, the almost daily supervision of diet, and constant careful examinations of the urine and heart. In connection with the treatment of obesity it was of the greatest importance to recognise whether it was of the inherited, or whether of the acquired form. In many cases which he had seen the mistake had been made of ordering one routine method of treatment without recognising whether it was a case of obesity which was acquired, or whether it was inherited. He agreed with much that Sir Lauder Brunton had said, and especially that water should not be taken at meals. The restriction of the dietary to meat alone, as in the Salisbury treatment, he thought dangerous, unless in very exceptional cases. Certainly it should not be adopted where there was any indication of albuminuria, any cardiac degeneration, or any valvular lesion; in such cases he thought it would be almost criminal to adopt it. We had employed a modified diet for obesity which in many cases was extremely successful. In that, the breakfast consisted of some form of either grilled white fish or grilled fowl, occasionally varied with a slice or two of tongue, one or two rusks, and weak tea. For lunch or dinner, grilled steak, green vegetables, salad, or lettuce, or watercress dressed with vinegar, a rusk, and the meal to finish up with an apple or a banana. With the grilled forms of fish and meat no sauces were allowed, but pure mushroom ketchup could be used. The most important article of fluid diet was hot water, and there again

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he was in accord with the advice to give it between meals. It should be taken freely one or one and a half hours before meals. He disagreed with the advice sometimes given to obese patients to drink as little water as possible. With regard to thyroid extract, he agreed with Dr. Burney Yeo, and would not countenance its administration if he thought it would permit an excessive indulgence in diet. But, given patients who were strong enough, he confessed he had seen very favourable results from its administration, conjoined with a suitable dietary. It should be given in carefully moderated doses, always beginning with a small one, the condition of the kidneys and the heart at the same time being carefully watched.

The PRESIDENT expressed to Sir Lauder Brunton, on behalf of the Fellows, the thanks of the Society for initiating such an interesting discussion. One could not but feel charmed, and at the same time envious of his great power of lucid exposition on such an abstruse subject, and his ability to embody in the remarks so much real scientific knowledge. Perhaps he (Dr. Allchin) might be allowed to express his regret that the opener had so misused his scientific imagination as to refer in the way he did to himself. Notwithstanding the extensive survey of the subject he had taken, Sir Lauder had left certain crevices in his remarks, which had been ably filled in, in great measure, by those who had followed him. Not the least interesting feature of the discussion was the fact that they had been able to pass an evening in the consideration of a subject from its scientific and ætiological side, as well as from its practical one, without any reference to the eternal microbe. They had been able to consider what, after all, was a problem of malnutrition, by which one meant that range of changes which take place in the organism, in virtue of which the structural integrity of the organism is maintained, and its functional manifestations rendered possible; the expression of the interaction which takes place between the organism on the one hand, and its environment on the other. With such a conception one must see there was a very wide range of possible conditions which might bring about that perversion of nutrition which they had been considering. Following that, of necessity, was the very wide range in the application of therapeutic measures, which justified the claiming of success for most varied modes of treatment. One could not help saying that the subject was characterised by considerable uncertainty, and that had been accentuated by the speeches that evening. To illustrate that uncertainty, one found a condition of obesity following on a state of over-indulgence in food, *i.e.*, in the amount of food, which, he confessed, he did not think was so frequent as was often supposed. But few of the speakers had referred to that class of person who got fat upon a very small amount of food. At two opposite conditions as regards quantity of food a very similar condition might result—namely, the production of corpulence. But the force of that was much diminished when one remembered that those conditions might equally exist without the production of corpulence, and might even exist with great leanness. Many very thin people ate a great deal, and, on the contrary, some thin people ate very little. There were also great differences in the conditions of the associated mental states. One saw, on the one hand, a great liability to the production of excessive fat in that class of person of which the cretin was an example; and in not a few defective congenital brain power was associated with the considerable accumulation of fat, notwithstanding that there might be nothing unusual or excessive in the amount of food taken, or any

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deficiency in the amount of exercise. Another illustration of the uncertainty which he had mentioned was the undoubted success which attended some one or other of those modes of treatment which Dr. Yeo had so ably dwelt upon, while there were many cases which would not yield to treatment at all. In endeavouring to find some explanation for that essential variability, and endeavouring to estimate the relative value of the different factors, one must realise the influence of heredity, which Dr. Yeo said operated to the extent of 50 per cent., that tendency on the part of the organism so to misuse the material supplied to it that it started on life with a vice of metabolism in the direction of the production of a large amount of fat in the working of the organism itself.

On coming to those conditions which were extrinsic, one was struck by the circumstance that Sir Lauder Brunton dwelt upon, namely, that the material of fat was obtainable from each of the three groups of food stuffs—proteids, fats, and carbo-hydrates, and probably more importance was attached to carbo-hydrates than to fats, because of the obvious circumstance that fats in the bulk formed a very small proportion of food in the actual diet, whilst carbo-hydrates constituted the bulk thereof. It was in dealing with carbo-hydrates that one had the widest opportunity for the treatment of the condition. But in the light of that very suggestive comparison by Dr. Burney Yeo of such cases of obesity with mild and severe cases of diabetes, he thought one might go a step further, and in endeavouring to trace the formation of fat on the part of the individual to ascertain how far it was obtained directly from the ingesta, and when it began to be formed from the structural nitrogenous material of the tissues themselves, as happened in the severe cases of diabetes. When the tissues of the body structure began to supply sugar, one had to deal with a different condition of diabetes altogether to those where the sugar was derived only from the intake of carbo-hydrates or sugar.

Another circumstance which had not been alluded to as a factor in the causation of obesity was that, however little might be known about it, there was a trophic influence exerted by the nervous system in the production of fat. This was in some measure shown in the peculiar condition of localised obesity known as lipomata, associated with severe pain, a condition which was also known as adiposis dolorosa, where masses of fatty material existed in certain parts of the body, preferentially in some rather than in others. Medical men could not shut their eyes to the opposite condition, namely, that of anorexia nervosa—that condition of extreme emaciation which was far beyond what could be explained by considerations of diet. He did not think such patients starved so often as was supposed. But even assuming a very small intake, they wasted a good deal more than healthy people would if they were to refrain from food to the extent those patients did. There was an associated perversion of their metabolism undoubtedly, through some flaw in the nervous control.

He was interested to hear Sir Lauder refer in the way he did—because it was in that direction one should look for possible information with regard to perverted metabolism—to what was expressed by the term internal secretion, or, at all events, some mutual influence exerted by the tissues on one another. Reference had been made, in support of that idea, to the influence which conditions of the sexual organs exerted. Young persons, even children, who had a tendency to be obese, were frequently defective or backward in their sexual development altogether, and a similar condition existed to that produced by castration in the direction of the laying on of fat. He thought physicians should not

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be altogether satisfied that they had got to the bottom of the matter by simply classifying the cases into those which were due to defective oxidation on the one hand, or to excessive ingestion on the other. That was probably true as far as it went, but there was very much behind it. Nevertheless, it was a problem of nutrition, upon which one awaited knowledge of the chemistry of the tissues. Until that knowledge was forthcoming there would be defective knowledge on the matter. But still it seemed desirable to even formulate the question, while admitting one's inability to answer it altogether, for such a step cleared the way, and showed the probable direction in which the extension of knowledge on the subject would take place.

Sir LAUDER BRUNTON founded his justification for his reference to the President on the philosophic speech which that gentleman had just delivered, which showed that the remark he made in his introduction was not a matter of unscientific imagination, but of actual fact. At that late hour he would not make any further remarks. He already felt that, as the speeches had all been so clear, it would have been a great advantage if his own observations had been shorter. He thanked the Fellows and visitors most heartily for their kind attention and criticisms.

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February 17th, March 3rd and 17th, 1902.

THE LETTSOMIAN LECTURES: ON CERTAIN DISEASES OF THE BLOOD-VESSELS.

By A. Pearce Gould, M.S. Lond., F.R.C.S. Eng.

LECTURE I.

Varicose Veins: Their Nature and Importance, and the Value of Trendelenburg's Operation.

Mr. President and Gentlemen,—My first and most agreeable duty is to thank the Council of the Society for the honour which they have conferred upon me in appointing me to deliver the Lettsomian Lectures. Would that I could make a worthier response. I can only ask that the kindness which prompted the invitation may be extended to me now as I offer a contribution which I know is not worthy either of this Society or of my predecessors in this office.

I believe that it is generally desired that the subjects of the Lettsomian Lectures shall be as practical as possible—that they shall deal with affections which perhaps often come under the notice of the Fellows of the Society, and in that belief I propose to devote this lecture to a consideration of certain points in connection with varicose veins. I will not attempt to give you an exhaustive account of this affection, or an analysis of the views that have been held in regard to the causes, the consequences, or the best treatment of varix. I will limit myself to a brief discussion of those features of the disease which have chiefly impressed themselves upon me, and will then submit to you the results of a somewhat extended trial of the particular method of treatment which was first employed by Trendelenburg in 1891, and which bears his name.

The Nature of Varicose Veins.

Two views of the nature of varicose veins are commonly held to-day. One regards them as the result of the yielding of the veins to an increased intravenous pressure due to an obstruction to the flow of blood up to and through the heart. In support of

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this much can be said. It may be pointed out that varix is met with most often in the veins of the lower limbs and left spermatic cord, and in those veins which are least well supported externally, the subcutaneous veins—that many of the subjects of them are following occupations which necessitate long standing or walking, or are suffering, or have suffered, from some obvious cause of venous pressure, such as the enlarged uterus of pregnancy. The relief that is sometimes afforded by external support to the veins is another fact that may be adduced in support of this theory. According to the other view varicose veins are to be regarded rather as venous overgrowths, a superabundant development of venous tissue—venous angeioma. In trying to decide between these two views the following facts and considerations have led me to reject the obstruction theory.

The Age at which Varices are first noticed.—In the great majority of cases varicose veins make their appearance before the growth of the individual is complete. They are one of the diseases of immature development. Out of 50 consecutive cases of varicose veins of the lower limbs collected for another purpose, but in which this point was noted, I found that 41 patients observed their varices before the age of 25 years, and only nine after that age. When we remember how unobservant patients, especially hospital patients, are of the date of onset of painless affections, and how constantly we have to discount their statements on this point, we may be sure that these figures represent less than the truth. Among the 41 cases, the age at which the varices were first noticed was given as follows :—

1 at 8 years.	2 at 18 years.
1 „ 11 „	3 „ 19 „
1 „ 13 „	4 „ 20 „
1 „ 14 „	1 „ 21 „
2 „ 15 „	5 „ 22 „
7 „ 16 „	2 „ 23 „
3 „ 17 „	6 „ 24 „

One patient said that she had had the enlarged veins "since she was a girl," and another said that "the veins had been enlarged as long as she could remember."

It is quite certain that puberty and adolescence are not the period of life in which venous obstruction predominates, nor are they the

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time of life at which strain is liable to tell upon the integrity of the vessel walls.

The General Condition of the Tissues of the Subjects of Varix.—I am sure that we must all have been struck with the general condition of good, even robust, health of the large majority of patients when varix is first discovered. How often it happens that the disease is only discovered when a typically healthy youth presents himself for acceptance in some branch of His Majesty's service.

The Conspicuous Absence in Cases of Varix of the Usual Symptoms and Consequences of Venous Obstruction.—This is another noteworthy point. We have many opportunities of seeing the effects of venous obstruction—œdema, lividity, and impairment of function. In the great majority of cases of varix these symptoms are all absent. I have seen the most extreme degree of varicose veins which had persisted for many years without any, even the slightest, alteration of the skin or subcutaneous tissue—neither œdema, induration, nor any loss of function—and such must be the experience of you all. But when we come to study other cases in which there is undoubted venous obstruction, how grave and striking these symptoms always are. Even in such a simple case as a slightly ill-applied bandage—just a very little impeding the venous return—how immediate is the discomfort, how quickly the whole member below becomes obviously congested, and how rapidly œdema supervenes. And in the graver cases of valvular derangement of the heart, or the growth of tumours, or plugging of a main vein, where the obstruction, even if it is slow and gradual in onset, is yet persistent, we are never at a loss to notice the grave effects upon the tissues when they are imperfectly drained of their venous blood.

The varicose veins that we know and are now thinking of are enlarged cutaneous and subcutaneous veins—parts of a very abundant, even superabundant, system of veins—and we are all aware of the fact that many of these veins can be removed or plugged with clot without any injurious effect upon the circulation of the limb, so many and free are the alternative channels which nature has provided for the return of the blood to the heart. But this fact has no bearing upon the point which we are now discussing, for the theory of obstruction always supposes that the obstacle to the free return of blood is one that acts upon the deep main veins and through them upon all the radicles communicating with them. And I submit to you that in uncomplicated cases of varicose

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veins there is, in the very great majority of cases, no evidence of general venous obstruction in the affected part at the onset of the disease—evidence which is not obscure or difficult to recognise and appreciate, but which stares us in the face in all cases of undoubted obstruction in deep veins. It is an error to accept the enlargement of superficial veins as evidence of obstruction in the main channels into which they open. The evidence of this obstruction is to be sought in general venous engorgement of the part, followed by œdema and its consequences.

The Result of Excision of Varicose Veins and also of Ligature of the Main Superficial Vein into which they empty their contents.—If the cause of a given varix is some deep obstruction, it is clear that to excise the varix is the most irrational procedure, and could in no case be fraught with good. Or to cause a further obstruction to the venous return by a ligature of the main superficial vein through which the varix is emptying its blood must, of course, exaggerate the evil and cannot possibly mend matters. And yet there is no clinical fact better attested than the success of both of these measures. I think that we have here a decisive refutation of the obstruction theory of varicose veins, and if anything could add to its force it would be the disastrous results that are obtained when an incautious surgeon operates upon veins which are enlarged as the result of obvious undoubted central obstruction—a femoral thrombosis, for instance.

The Influence of Pregnancy.—Women often tell us that they had not been troubled by enlarged veins until during or after pregnancy. It is, indeed, commonly held that pregnancy plays an important *rôle* in the etiology of varix. Before we can rightly judge of its importance, however, there are several other facts to be borne in mind. At the outset we must carefully distinguish between conditions causing varix and conditions leading to pain in varix. Over and over again observant women have told me that they had noticed their enlarged veins in early life before marriage, but that they had *never been troubled by them until they were pregnant.* If one is careful to press this distinction, it soon becomes obvious that while pregnancy is often a cause of pain in varix it is not very often the cause of varix. It is also noteworthy that it is not uncommon for a woman to say that her veins were only noticed to be enlarged when she "got up after her confinement," or even later, when she "weaned the baby." While admitting, of course, that the pregnant

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uterus may be, and often is, a cause of pressure upon the common iliac veins or vena cava, and so acts as an impediment to the venous circulation in the lower limbs, it is to be remembered that, quite apart from any pressure-effects, pregnancy produces in some women quite remarkable vaso-motor disturbances, and it is an error to conclude that all venous engorgements must of necessity result from the mechanical pressure of the enlarged uterus. In this connection it is important to notice that many women complain of pain in their varicose veins at the very commencement of pregnancy, before the uterus has risen high enough to press upon either common iliac vein, and these women not infrequently tell us that those pains abate in the later months of pregnancy, when the pressure upon the veins must be at its maximum. I believe, too, that it is a fact that large ovarian and utreine tumours are less often attended with troublesome varices of the lower limbs than in pregnancy—a fact (if it be a fact) which surely points to some other factor as well as pressure upon the main veins coming into play in the enlarged and painful varices of pregnancy.

The Sex-incidence of Varix.—But if we look at the influence of pregnancy from another point of view altogether, we shall, I think, see still further reason to believe that its influence in causing varicose veins has been over-estimated. When we consider the frequency of pregnancy in the female sex, and the great abdominal distension and pressure that it causes in the later months, I think that we shall be bound to admit that there is no condition, or even group of conditions, in the male sex that can possibly equal the effects of pregnancy in embarrassing the venous circulation of the lower limbs. And therefore on the obstruction theory we should expect to find varices of the lower extremities much more common in women than in men. But what are the facts? Sir William Bennett's statistics show the incidence to be about equal in the two sexes. I have gone over my notes of all the cases that I have seen in private practice, and to them I have added all the cases in my hospital practice for the last six years, excluding cases of varicocele —they number 245. Of these patients 153 were men and 92 were women, a proportion of rather more than three males to two females. At the hospital I have more female than male beds, and I am unaware of any reason why my experience of the frequency of this affection in the two sexes should not represent the fact. If so, I think that we must conclude from this survey of the sex-incidence

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of the disease that pregnancy cannot play an important part in causing varix. If I had included all my cases of varicocele during the same period the sex-incidence would, of course, have shown a much greater preponderance in men.

But if we reject this first theory what grounds are there for adopting the other—the view that varicose veins are venous overgrowths—venous angeioma? Before answering that question it may be well to ask another: Is there any evidence that a growth of veins—a highly-specialised structure—ever occurs in adolescent or adult life? We have all seen cases of the development of arterial angeioma or aneurysm by anastomosis, and that forms a fair analogy; and we are very familiar with venous angeioma as a congenital growth which if neglected may continue to grow after birth and assume even large proportions. We cannot fail, too, to be impressed with the apparent superabundance of veins in the normal condition. Nature appears to be very prodigal in the supply of veins, and nowhere more so than in the primary veins of the lower limbs and their tributaries. Large numbers of veins may be obliterated without any interference with the capillary outflow. These facts would prepare us to observe a growth of veins in adults, and I think cases like the following are conclusive evidence that such a growth does occur.

Case 1.—In October, 1895, an agricultural labourer, 46 years of age, was admitted to hospital under my care suffering from extensive varices in the lower third of the left thigh and in the left leg. These caused him so much pain that he was unable to continue at his work, even although he wore an elastic stocking or a rubber bandage. I excised about 1½ inches of the internal saphena vein above the highest varix and a little below the middle of the thigh. In a fortnight the man returned to his work, and for two years he was able to work in perfect comfort, the varices having in the meantime shrunk and being always lax. At the end of two years he began to complain of the old pain, and I found that the varices were fuller and tenser. On looking at the thigh I found that the saphena vein was varicose above the scar of my operation, and close alongside the scar a large vein could be plainly seen and felt passing from the saphena vein above the ligature to the vein below, and this accounted for the return of the man's pain, &c.

I have seen other similar cases where, after I have cut out a piece of the saphena vein in the middle of the thigh, either a single large vein or a number of veins have developed in the scar and have formed a new and free communication between the divided and separated ends of the saphena vein. The other day, when examining some of my old hospital patients on whom I had

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performed Trendelenburg's operation—excision of the upper end of the internal saphena vein—I came upon such a case (No. 16 in the table of cases).

CASE 2.—The patient was a man, 41 years of age, upon whom I operated in July, 1900. He had then rather large varices in the left leg and an ulcer over the inner malleolus. He left the hospital with the operation wound and the ulcer soundly healed. There is now a large varix in his groin which communicates with the upper end of the lower part of the divided saphena vein, passes round the outer edge of the scar, and seems to enter the stump of the saphena vein where it opens into the femoral vein.

Another case (No. 8 in the table of cases) occurred in the person of a member of our profession.

CASE 3.—The patient himself thus describes the case:—"At the age of 19—in 1894—I had tortuous veins on the inner side and back of both calves and a prominent internal saphena vein in each thigh, in which could be felt a thrill on coughing. At the left saphenous opening was a slight bulging of the vein. Operation in 1894 by Mr. X—excision of a portion of the internal saphena vein in the upper part of the thigh, but I think about an inch or more was left between the ligature and the saphenous opening. This relieved me, and the aching pains I used to get went away. Then the veins began to get big again in the calf, and in the site of the operation in the left thigh a large mass of veins began to grow which were bigger than I had had before anywhere. These Mr. X excised for me in 1896, at the same time excising most of the tortuous or prominent veins in the calf of each leg. For about three years I kept very free from any discomfort from my veins, but in the summer of 1899 I began to feel them and I had sudden twinges of pain in them when standing in the wards and theatre. They (the varices) had then recurred in the left calf, the right remaining well." This gentleman then consulted me, and I found the scar in the upper part of the left thigh occupied by quite a close congeries of large veins which communicated freely with the two ends of the saphena vein. I operated in October, 1899, and removed these veins and tied off the upper end of the saphena vein as close as possible to the common femoral trunk. He adds in the letter which I have quoted from above, "I have been very comfortable since and have no pain in them now." In this case, then, new veins of considerable size were seen to develop twice over in a scar in the thigh, and each time these veins acted as a free channel of communication between the divided ends of the internal saphena trunk.

I think that such cases may be fairly quoted as demonstrating the occasional development in the adult of what may be classed as "venous angeioma." Admitting the possibility of the development of a venous angeioma in the adult, what evidence is there to support the view that varix is a form of venous angeioma?

1. In the first place I would point to the fact that we occasionally meet with cases in which we have a *combination of undoubted venous nævus and varix*, and other cases in which the condition might

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almost equally well be designated by either of these terms. Several years ago I showed to this Society a young woman who presented a most remarkable appearance. The left half of her body from head to foot was the seat of a very extensive diffused angeioma—in great part it was a capillary nævus, part of it was rather of the character of a venous nævus, and yet other parts were indistinguishable from ordinary varix. This was a congenital malformation or error of development, but it suggested to me very powerfully the similarity of varix and venous angeioma. This month I had in the Middlesex Hospital a man with a large venous nævus of the scrotum which occupied the whole of the scrotal raphe and extended along the under surface of the sheath of the penis, and also back into the perineum. I excised this growth, and in doing so exposed and removed, in the subcutaneous scrotal tissues, veins which I could not distinguish from ordinary varices. Further, this man had on the inner edge of his left buttock an area of subcutaneous fascia about 2 inches in diameter occupied by dilated veins or venous spaces, and on the lower part of the thigh and the leg of the same side were some quite unmistakable varicose veins. Two or three weeks ago I saw a young woman on whom I had performed Trendelenburg's operation some time before. Her thigh presents a truly remarkable appearance. She is well nourished, with abundance of subcutaneous fat, and in this fat the varicose veins are so numerous that they give a blue tint to the whole limb, and I think that it is as reasonable to regard the condition as a very diffused nævus as it is to speak of it as a very extensive varix. Only this week I saw a patient, 50 years of age, who has what she calls a "wine-and-water mark" covering the upper two-thirds of the right leg and nearly the whole of the thigh, on the outer side extending up to the groin. The subcutaneous tissue is occupied by a close network of dilated veins which project on the surface when very large, and give to the whole part a blue colour. The veins are indistinguishable from ordinary varices, but the condition is peculiar in having been present since birth. Many years ago I saw among my out-patients at the Westminster Hospital an old man of 80 years of age whose right leg from knee to ankle was closely covered by a congeries of large veins, many of them of the size of a woman's little finger, which were so numerous that nowhere could a shilling have been placed on the leg without resting on one of these veins. The old man told us that his leg had been like that for 60 years and

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had never given him any trouble; he came to the hospital for quite another affection. More common, and, I think, more striking than these cases, are those others of varix involving the minute veins of the skin. We often see a kind of splash of this condition just above the knee, sometimes superficial to a dilated saphena vein. How exactly it resembles the mildest form of nævus of the skin, or the edge of a nævus undergoing spontaneous cure! Indeed, I think that I have seen every grade between the undoubted congenital venous angeioma and the most uncompromising acquired varicose vein, and this fact has certainly borne in upon me the essential oneness of the two conditions.

2. Notice again the *capricious irregular distribution of varices.* In one case it is the whole saphena vein from groin to instep that is enlarged, no one of its tributaries being involved. In another case it is merely a short section of this vein that is varicose. In a third case the saphena trunk and several of its main tributaries are dilated. In a fourth case the disease involves only some second or third-rate tributary and leaves the main vein unaffected, or a large varix may be found lying over the normal vein. Large varices are not uncommonly seen in situations where no vein of any size is normally present—*e.g.*, across the front of the patella. Lastly, in a few cases the enlargement appears to involve an enormous number of veins in the whole subcutaneous tissues of the leg or thigh almost uniformly, although the trunks may not be specially enlarged—and yet there is no sign of any impairment in the venous drainage of the tissues. This extreme irregularity and variation in the seat and extent of the affection seem to me to mark it out as an over-development of the venous tissue—errors of development often showing such caprice.

3. *The early age at which varix usually develops* is another interesting fact bearing upon its nature, and eminently consistent with the developmental theory.

4. Further evidence pointing to the same conclusion lies in the fact that *varicose veins seem to be distinctly hereditary.* It is very difficult to express this with anything like mathematical accuracy, as a large proportion of patients are unable to give any information about varices in other members of their family. It is not an obvious deformity or defect that cannot be hid—a sister is not likely to know of a brother's varicocele, or a brother of a sister's varix unless it causes serious inconvenience. But in spite of this difficulty in

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getting anything like full family histories on the point, it is surprising how often patients tell us of other members of their family who have varicose veins too. The young medical man whose letter I have just quoted mentions in that letter that his father and one of his two brothers have varicose veins. Two days ago I operated upon the daughter of a medical man for varix in each lower limb, whose father also suffers from varicose veins. A gentleman whom I have seen on two or three occasions for very large varicose veins which he has had all his adult life, and which have in no way interfered with a most active life, including such sport as deer-stalking, mentioned on his last visit that his eldest-son—a youth in his teens—was just developing varices in his thighs and legs. I might, indeed, quote from my note-book case after case illustrating the same fact. The very last case of varicose veins I saw was that of a young man who told me that his maternal grandmother, his father, and his two brothers had varicose veins in the legs—he did not know about his sisters. Out of 102 cases of varix of the limbs, collected for another purpose, I find that the disease was known by 52 patients to be present in some member or members of their families—more than 50 per cent. In the table of cases of Trendelenburg's operation I have put this detail in where I could, and if you will run your eye over the table, I think you cannot fail to be struck by the frequency with which the entry occurs. We know nothing of the real nature of hereditary influence, but we notice that it seems to show itself more often in errors of development, whether by way of excess, or defect, or variation, than in any other way.

5. *The striking innocuousness of varicose veins* in the great majority of cases also, I submit, supports the same view of their nature. An extreme degree of varix persisting for many years is by no means incompatible with unimpaired function and structure of the parts draining into the enlarged veins, and the varicose veins themselves will often persist throughout life without undergoing any secondary or further changes. This *stability* of varicose veins is, I think, an important point. We often find them persisting quite unaltered for many years, and they are certainly less troublesome in the later than in the earlier years of adult life. It seems to me that this fact is more compatible with the view that they are an exaggeration of a normal physiological structure than the result of a pathological process.

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6. The last point I would mention is the *minute structure of a varicose vein.* I believe that all the normal constituents of a vein are found in a varix, and are usually increased in amount. It is often stated that the thick walls of a varix—and they may be very thick—are composed almost entirely of fibrous tissue. I have examined a good many, and that has not been my experience: an increase of the muscular tissue is as notable as the increase of fibrous tissue, and endothelial hyperplasia is sometimes a marked feature.

These, then, are the reasons which seem to me to show that varices are essentially the result of an over-development of venous tissue closely allied to, if not identical with, angeioma.

But associated with this growth of venous tissue there is very usually a failure in the valve apparatus which allows of regurgitation of blood from the main deep veins into the superficial veins. Since reading Trendelenburg's paper I have investigated this point in every case of varix of the lower limbs that has come under my observation, and I have found that regurgitation into the superficial veins is an almost constant phenomenon. The observation is made thus. Having examined the patient in the standing position, he is asked to lie down on his back, and the limb to be tested is raised to the vertical and the varices are completely emptied by gentle centripetal pressure. The thumb or forefinger is now placed in the groin so as to compress the upper end of the internal saphena vein, and the patient is told to rise. It will be found that almost without exception the varices will remain lax and almost empty so long as the compression is maintained upon the main vein above, but that they fill out at once from above when the compression is removed. The regurgitation of blood into the superficial veins is sometimes most striking and obvious—in other cases it is less free, but I have rarely failed to demonstrate it in any well-developed case of varix. In some instances I have found that it has occurred from the popliteal into the external saphena vein instead of, or as well as, from the common femoral vein into the internal saphena vein. In one case I found that the back flow occurred through a small vein passing up the thigh just internal to the line of the anterior iliac spine as well as into the internal saphena vein. This regurgitation is not a normal condition. At my request Dr. H. Campbell Thomson has made observations on this point in the *post-mortem* room at the Middlesex Hospital, and he has entirely

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failed to inject fluid from the external iliac vein into the internal saphena vein, owing to the efficiency of the valves at the mouth of the superficial vein. He has made for me some preparations showing these valves. There is a double valve at the mouth of the saphena, the cusps being very unequal in size; 2 inches lower down another pair of cusps is found, and so on down the vein. In the normal state these valves are quite efficient. I am convinced that the connection between insufficiency of these valves and varix is close and important, and I must confess that even after reading Trendelenburg's paper I was not prepared to find the regurgitation so nearly constant or so marked as it is.

Before passing on to speak of the treatment of varix, I should like to say just a word about the importance of the affection. I am certain that the public have a very exaggerated notion of the gravity of varicose veins, and one comes across certain neurotic individuals whose life is made miserable by the constant anticipation of evils which never arise.

The effect of varices upon the nutrition of the part, whether the leg or the testicle, is, I believe, very small, if indeed it be at all perceptible. Certain I am that many persons carry about in their legs and thighs for many years very large and numerous varicose veins, unalleviated by any external pressure or support, without suffering in any way at all—the skin remains elastic and supple, the subcutaneous tissue is free from œdema or induration, and the surface is proof against eczema or ulcer. The troublesome ulcer of the lower third of the leg with thickened edges and unhealthy base—so slow in healing, often so painful—the ulcer that I think has been nicknamed "varicose"—certainly occurs in splendid perfection in limbs devoid of a single varix, and its cause must be sought for elsewhere than in the veins.

The most frequent ill-effect of varices is a sense of fulness or weight in the part which may amount to severe aching in the distended veins. It is brought on by prolonged standing or walking, and comes on much sooner in hot climates. It occurs only in the upright position, and is at once relieved and quickly removed by recumbency. Some patients complain of a pricking or starting pain in the part. Neither the extent, nor the seat, nor the degree of the varix affords a measure or explanation of the suffering attributed to it. Many patients with well-marked and even extensive varices are able to undergo considerable fatigue

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without discomfort, while others, with apparently far less cause, make grievous complaint. The most striking instance of this that I have seen was a nervous, ill-nourished man, with a quite moderate development of varices in both legs. He described his sufferings from the veins as most severe, and sufficient to make his life miserable, and he gravely assured me that often when attending meetings of boards of directors in the City he had to retire into an adjoining room and to stand on his head "to let the blood out of his legs."

This is the only *frequent* ill-effect. Much less often we meet with thrombosis in a varix or started in one, and more rarely still a vein bursts or is ulcerated into and free venous hæmorrhage occurs. Where varicose veins do not trouble the patient he need rarely trouble about them. But a young woman with large varices is wise to have them treated before marriage and pregnancy, and young people of either sex should have them treated before going to live in a hot climate. Thrombosis I propose to speak of in a subsequent lecture.

Trendelenburg's Operation.

Believing that insufficiency of the valve at the opening of the internal saphena vein plays a great part in causing varix below, Trendelenburg, in 1891, proposed to treat the disease by division of the upper end of this vein between two ligatures. The operation has been extensively practised, but I believe that there is a wide divergence of opinion as to its value. For the last six years I have treated by Trendelenburg's method almost every case of varix in the lower limbs that has required operation, and as my cases up to the end of 1901 numbered 120, I hope that my results, so far as I can describe them, may be of service in leading to a correct estimate of the value of this treatment. The operation has the great advantage of being a simple one, rarely requiring more than five minutes for its performance. Gas and oxygen are often the only anæsthetic needed. The saphena vein can readily be found by remembering that it lies a finger's-breadth to the inner side of the common femoral artery, the pulsation of which can easily be felt in the groin. I prefer to expose the vein by an oblique incision from 1½ to 2 inches long parallel with the fold of the groin and having its centre over the vein. The vein is found lying between

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veins in the thigh are more and more constantly affected than are those in the leg.

Influence of the Operation upon Thrombosis.—Some of the patients operated upon had suffered before the operation from recurring thrombosis. It is certainly a most noteworthy point that the tendency to thrombosis seems to be very greatly lessened, if not entirely removed, by this operation. In support of this statement I should like to mention one or two cases:—

CASE 4.—The patient was a woman, 52 years of age, who had suffered very severely for many years from very extensive varices in both lower limbs. She had for years worn elastic bandages from the feet to the groins, and even with this support she was greatly hampered in her work as a nurse—"never any comfort, sitting, standing, or lying." Shortly before my operation in February, 1898, she had experienced four separate attacks of thrombosis, which had kept her confined to bed for a long period, and had greatly impaired her usefulness. She has had no thrombosis since the operation, she has lost all her pain, has been able to dispense with the elastic bandages on the thighs, and, as she puts it, she "hasn't felt so well for years" as she does now. She is, indeed, very enthusiastic in her estimate of the value of the operation to her.

CASE 5.—The patient was a healthy, robust man, 29 years of age, who was sent to me by Dr. F. W. Burton-Fanning, of Norwich. His case was a little peculiar, inasmuch as in addition to an enlarged internal saphena vein in the right thigh and numerous varices in the leg, there was an enlarged vein running up the front of the thigh and entering, apparently, the circumflex iliac vein. Pressure upon the saphena vein did not "control" the varices below unless this vein was also compressed. This patient, in addition to aching pains in the veins, had suffered from several slight attacks of thrombosis just before he consulted me. I ligatured and divided both of the veins in the thigh (No. 49), and the result has been a distinct diminution in the size of the saphena vein and its tributaries, but no such change in the other vein, which seems to be "fed" by a deep communication about the middle of the thigh. He is able to walk, to ride, to shoot, to cycle, and to play golf with comfort, and he has had fewer and slighter attacks of thrombosis, and they now seem to have ceased altogether.

CASE 6.—This case is No. 10 in the table. The patient, a woman, 28 years of age, had very frequent attacks of thrombosis—often every other month—up to the time of the operation. Since then—a period of two and a quarter years—she has had two "slight attacks" only, "very slight to what it used to be."

Where the varices are only "controlled" by pressure upon the external saphena vein as well as the internal, I now obliterate the upper end of each vein, and I think that I have succeeded in benefiting cases by this means which in my earlier experience did not reap the full benefit of the operation.

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TABLE OF CASES OF VARICOSE VEINS SHOWING THE RESULT OF TRENDELENDBURG'S OPERATION.

No.	Age.	Sex.	Date of Operation.	Condition before Operation.	Result.
1	31	F.	March, 1899.	Internal saphena vein was tied in the middle of the thigh on each side in 1896: after a period of relief the pains had recurred and the veins had enlarged again. Now the veins were tied in the groin.	Letter, January, 1902: "Since the last operation the veins have given me little pain; my legs have not swollen much, or prevented my doing a fair share of domestic duties."
2	18	M.	March, 1899.	Exceedingly large varices below the right knee; continuous dull, heavy pain in them. One brother had varicose veins.	Seen January, 1902. The veins were much smaller; there was no discomfort except a slight pain if he over-exerted himself. There was no new varix.
3	37	F.	May, 1899.	The veins were large but not painful; one burst and a great deal of blood was lost, and this led the patient to seek advice. Her mother had varicose veins.	Seen January, 1902. The veins were much smaller; they caused no trouble at all.
4	29	F.	May, 1899.	The patient had varicose veins in both legs for 10 years: they ached on walking about. Three sisters had varicose veins.	Letter, January, 1902: "The varicose veins are decidedly better; one leg is quite cured, but the other swells a little at times, but is seldom painful."
5	28	F.	June, 1899.	Varices in both legs and above the left knee. The patient could remember the varicose veins since the age of 11 years; they had not altered much up to the time of the operation. One brother had been operated on for varicose veins.	Seen January, 1902. The operation has done her decided good; she could walk much better; the legs were painful if she stood for two or three hours, but not so much as before.
6	26	F.	October, 1899.	Bunch of veins just below the knee; there were several smaller ones lower down; there was pain on standing.	Letter, January, 1902: "Has had no pain or discomfort since the operation."
7	20	M.	October, 1899, and March, 1901.	The patient had had varicose veins for four or five years; they were very painful and ached very much after a hard day's work. The right internal saphena vein was tied in 1899; the left internal saphena vein and the right external saphena vein were tied in 1901. His mother had varicose veins.	Seen January, 1902. The operation had done great good; the veins were cured. He was employed at a large city warehouse, and had no pain or discomfort.
8	24	M.	October, 1899.	Saphena vein in the thigh prominent from the groin to the knee, bulging at the saphenous opening; tortuous varices over each calf. Two previous operations. Recurrence of varix in the left calf with pain on standing. His father and one brother had varicose veins.	Letter, January, 1902: "Quite free from any discomfort; veins hardly visible when cool; after long standing, or getting hot, they show up."
9	21	F.	November, 1899.	Noticed veins seven years previously. Had a good deal of pain, which was getting worse. Both internal saphena veins were dilated and slightly tortuous. One sister had varicose veins.	Seen November 20th, 1900. Left internal saphena vein above the knee was large and varicose, veins below knee quite small; no aching. On the right side the veins had diminished in size considerably since operation; a little aching pain after prolonged standing.

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10	28	F.	November, 1899.	Very numerous varices scattered generally over both thighs and legs, giving the limbs quite a bluish colour. Subject to frequent attacks of thrombosis—as often as every other month. Her father had varicose veins.	Seen January, 1902. Since the operation she had had a slight thrombosis each autumn, "very slight to what it used to be."
11	27	M.	November, 1899	Extreme condition of varix in the right thigh and leg; slight on the left side. His father had varicose veins.	Seen January, 1902. The right leg was quite well; the old varices could be felt—shrunken, soft, and lax. The left leg was worse; there was a varix at the back of the calf opening into the external saphena vein which ought to have been tied.
12	33	M.	May, 1900.	Veins on the inner side of the thigh and legs were very swollen and tender. Excision of varices was performed at St. Thomas's Hospital at 19 years of age; others formed in the leg and thigh; thrombosis occurred in the thigh. His mother and one brother had varicose veins.	Seen January, 1902. Had no trouble in the legs after the operation; was passed for military service in South Africa, and was quite well there till September, 1901, when he suffered from a saddle-gall.
13	28	F.	June, 1900.	Noticed vein three years. A year previously the leg became much inflamed and very painful. Large vein ran from the knee to the ankle. One sister had varicose veins.	Letter, January, 1902: "Veins are quite the natural size and do not swell. I have not had any pain or discomfort since the operation."
14	32	F.	June, 1900.	Pain from enlarged veins in both legs for three years; lately much worse. There was some eczema of both legs. The veins of both legs were varicose. The left saphena was enlarged above the knee.	Letter, January, 1902: "Veins are now their natural size, not swollen, and are free from pain, and I now follow my occupation without any discomfort whatever from them."
15	27	M.	June, 1900.	Both internal saphena veins were enlarged and very tortuous from the saphenous opening to the foot. The patient had not had pain in them. The enlargement started in the calf and had spread upwards.	Letter, January, 1902: "Veins of left leg are normal. Those of my right leg have swollen slightly since the operation. The veins are not painful, and do not hinder me in my work."
16	41	M.	July, 1900.	Had pain from the veins for four years. Had an ulcer above the internal malleolus for the last two years. Was compelled to rest in bed occasionally for from two to three weeks at a time. Internal saphena in its whole length was varicose. Had large ulcer over the internal malleolus at the time of operation. The skin round it was eczematous. One brother had varicose veins.	Seen January, 1902. Leg did not trouble him now "in the least." There was no varix in the thigh; those in the leg were smaller. The scar of healed ulcer was quite sound. A considerable varix had developed below the operation scar and opened into the highest part of the internal saphena vein.
17	35	M.	July, 1900.	Veins enlarged for 16 years; they had been painful for the last few months. There was some eczema on the left leg. The internal saphena was large and prominent on both sides. There was great dilatation of the vein at the saphena opening on the left side. His father had varicose veins.	Seen January, 1902. He experienced marked relief for a year afterwards. There was no pain now in the right leg; the left leg pained him below the knee. The vein in the thigh was very small; below the knee were two varicosities much smaller than before.
18	25	F.	August, 1900.	Veins swollen and painful for the last three years. The veins were enlarged on the front of the right leg and the inner side of the left thigh. Other members of the family had varicose veins.	Letter, January, 1902: "My legs are very much better. The vein in the right leg is still a little swollen and rather painful periodically, but generally speaking I do not feel anything of it, and it is nothing like it was before the operation." The veins in the left thigh cause no trouble.

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TABLE OF CASES OF VARICOSE VEINS—*continued.*

No.	Age.	Sex.	Date of Operation.	Condition before Operation.	Result.
19	37	M.	August, 1900.	Varicose veins in the left leg "since a child"; nine years before operation a vein burst. In 1889 at the Italian Hospital three varices and a thrombus were excised. Lately the veins had been very painful. There were large blue swellings beneath the scars of former operations.	Seen January, 1902. A very good result. The veins were shrunk and lax. He said that he "occasionally has a pricking pain if he stands very much."
20	27	F.	August, 1900.	Was a nurse in hospital. Had varicose veins on both sides.	Letter, January, 1902: "Since the operation I have had no trouble with my legs: they do not swell, are not painful, and I can follow my occupation as a nurse with no discomfort, though the veins are still slightly enlarged."
21	65	F.	October, 1900.	Varicose veins "noticed when a girl." Had an ulcer occupying the lower third of the front of the left leg. There were numerous varices above it.	Letter, January, 1902: "My leg is quite well," no pain or swelling. The veins are their natural size. "I can do all my house work and take walks without discomfort."
22	19	M.	November, 1900.	Varicose veins noticed at eight years of age. Pain and the varicose condition had been getting worse during the last two years. The enlarged veins were situated on the back and outer side of the left calf. His mother had varicose veins.	Seen January, 1902. A very good result. The large varix just below the knee had disappeared; those over the inner side of the calf were much smaller and soft. "Never feels anything of it."
23	35	F.	November, 1900.	Had great irritation in the legs for the last five years. For the last two years had to rest repeatedly at work on account of pain. Large tortuous varices in both thighs and legs. Her mother had varicose veins.	Seen January, 1902. No pain in the limbs; was able to do her work better. Up to the time of operation the veins were enlarging; since then there had been no enlargement. There were numerous varices in both thighs and legs, but much less tense than before.
24	39	M.	January, 1901.	The patient was rejected for the navy at 16 years of age on account of varicose veins. In October, 1896, a vein burst and an ulcer followed. Since then two others had appeared. He had dull aching in both legs after any exertion. He had varices in both legs and thighs. His mother had varicose veins.	Seen January, 1902. No varices in the thighs; those in the legs were smaller and quite lax; he experienced no trouble from them.
25	28	F.	January, 1901.	Had varices both sides for eight years. There was increasing pain in the right knee and thigh for the last six weeks. The internal saphena in the right leg and thigh and in the left leg was enlarged. Her mother had varicose veins.	Seen January, 1902. The veins were much smaller.
26	26	F.	January, 1901.	Veins noticed to enlarge eight years previously during her first pregnancy. On the left leg were two small ulcers. Above the left knee there was a sacculus on the internal saphena vein of the size of a nut. The vein was enlarged down to 2 inches below the knee. Her mother had varicose veins.	Seen January, 1902. Scars of ulcers were quite sound. "No pain at all in the leg now." She felt "much better for the operation."

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27	38	F.	February, 1901.	In March, 1896, the right internal saphena vein was tied above the knee; there was great relief for two years, then a return of the varices in both legs "controlled" by pressure on the internal saphena vein.	Seen January, 1902. "Left leg is always in pain. Right leg aches terribly at times; just the same as before." On the right leg numerous varices had developed in the scar and the varices below were as bad as ever. In the left leg the veins had shrunk, and the pain complained of was not due to the veins; it was bad when she rested.
28	40	F.	February, 1901.	The veins were noticed 18 years previously during her first pregnancy; they were relieved after labour, but enlarged during each pregnancy. Twelve months ago an ulcer appeared on the left leg. For the last 12 months pain day and night had been very severe. The internal saphena veins of the thighs and legs were varicose, also the veins of the feet. Her mother and two brothers had varicose veins.	Seen January, 1902. "No trouble since the operation." Ulcer soundly healed; the veins are smaller.
29	18	M.	February, 1901.	Enlarged veins were noticed for four months. He came for treatment on account of the pain. The left internal saphena was enlarged in the thigh and leg.	Seen January, 1902. Now no pain in the left leg, the one operated on. Complained of pain in the right internal saphena vein which had not been operated on.
30	35	M.	April, 1901.	Had great pain for several weeks before the operation, so that he could not stand upright. An ulcer had existed for six weeks. The left internal saphena was enlarged in whole length above the ulcer. His mother and sister had varicose veins.	Seen January, 1902. Veins were a little smaller; there was no pain.
31	25	F.	April, 1901.	The whole leg below the knee was thickly studded with varices giving it a bluish look. Her mother had had varicose veins.	Seen January, 1902. She did not think there was much difference since the operation.
32	27	F.	April, 1901.	Noticed veins for 10 years. Got rapidly worse during the last three months. There were groups of varices on the inner side of the knee, joining others on the outer side of the leg. There was another group on the inner and posterior aspect of the calf and inner side of the ankle.	Letter, January, 1902: "No benefit from operation. The veins swell as much as they did before directly I stand on my legs, and also are very painful at times."
33	17	F.	May, 1901.	Varicose veins for two years. The leg was painful both on standing and sitting. The internal saphena was enlarged in the right thigh and both legs.	Seen January, 1902. The veins were quite small; there was no œdema or other complication. She complained of "pain and shaking in the legs"—a neurosis.
34	29	M.	May, 1901.	Started about five years previously. The veins on the inner side of the right thigh and leg were much swollen and tortuous, also on the inner side of the left thigh. His father, mother, and two brothers had varicose veins.	Letter, January, 1902: "The operation has been attended with perfect success. Since about six weeks after the operation there has been no pain or swelling, the veins are reduced to their natural size, and I am able to attend to my usual occupation without the slightest inconvenience or discomfort."
35	22	M.	May, 1901.	Painful on standing	Letter, January, 1902: "The veins still swell. The veins are not painful, and I am able to follow my occupation without discomfort from them."

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TABLE OF CASES OF VARICOSE VEINS—*continued.*

No.	Age.	Sex.	Date of Operation.	Condition before Operation.	Result.
36	38	F.	June, 1901.	The patient came to hospital on account of pain. The veins had been enlarged for 22 years. There were varices on the inner side of the left leg, also on the inner side of the right leg and thigh and the outer side of the right foot. Her father had varicose veins.	Seen January, 1902. Quite free from pain.
37	31	F.	June, 1901.	The veins were painful. There was a large varix on the inner side of the left leg.	Letter, January, 1902: "I am able to get up and down stairs better than I have done for the last 10 years. There is no pain. The veins still swell."
38	26	M.	July, 1901.	The leg ached after standing; the patient was afraid of rupturing a vein when at work.	Letter, January, 1902: "Legs quite well. I still follow my usual occupation, which is that of a coppersmith, and necessitates a great strain on the legs. The veins are their natural size and are perfectly smooth, and I feel no discomfort whatever from them."
39	17	M.	October, 1901.	Veins enlarging for one year. Ached slightly after work. Trendelenburg's operation was done on both sides and at the upper end of the short saphena of the right side.	Seen January, 1902. The veins were smaller. He had not felt any inconvenience in them since operation.
40	32	F.	October, 1901.	Varix at the upper end of the internal saphena vein; ulcer on the right leg.	Seen January, 1902. She had lost all pain. The scar of ulcer was quite sound: there was no œdema.
41	22	M.	October, 1901.	The veins caused slight irritation at night	Seen January, 1902. The veins were much smaller. He had no inconvenience from them.
42	39	M.	November, 1899.	A very neurotic man. Varix was first noticed at 18 years of age, and had been steadily increasing ever since. The right internal saphena vein was varicose in the lower quarter of the thigh and below the knee. In the left leg was a bunch of varices communicating with both the internal and external saphena veins and other varices lower down. Both the internal and the left external saphena veins were tied.	Seen May, 1900. All the varices were decidedly better. He complained now of pain on standing, which was relieved by raising the legs. This pain started in the right buttock, passed down the back of the thigh to the knee, to the heel, and on to the base of the fifth metatarsal bone. There was tenderness on pressing over the great sciatic nerve.
43	41	F.	February, 1900.	Varices in each leg which were only controlled when the external and internal saphena veins were compressed. The patient had worn elastic stockings for years, and even with them had great pain on standing and walking. Felt "quite well down to her groins." Both the internal and the external saphena veins were tied.	Letter, February, 1902: "I have no pain; one leg is perfectly cured, and the other just shows the vein. I am often glad that I went through the operation."

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44	59	M.	April, 1900.	Numerous large varices in the left leg involving the internal saphena vein and its branches; the main vein was enlarged up to the lower half of the thigh and at the saphenous opening. When the internal saphena vein was compressed two small varices in the calf partly filled out; when the external saphena vein was also compressed all the varices were kept empty. Both the saphena veins were tied.	Letter, February, 1902: "I have never had a moment's trouble since my operation, and my legs are in perfect condition."
45	53	F.	March, 1900.	Many varices in the left leg and thigh which ached greatly when she stood. Had had one attack of thrombosis.	Letter, February, 1902: "Veins are only slightly swollen. No pain on standing or walking."
46	30	F.	April, 1900.	The right internal saphena vein formed many tortuous loops up the leg from the foot to just above the knee. There was aching pain. Operation was advised in view of approaching marriage.	Letter, February, 1902: "No pain or trouble. Veins smaller. Has had one child and one miscarriage. No trouble during pregnancies."
47	39	F.	July, 1900.	Varices in the right thigh. There were much aching and twitching pain on standing and walking.	Seen February, 1902. The patient was very much better. There was no pain now; she was able to do her work as a nurse without discomfort. The veins were smaller.
48	44	M.	May, 1901.	Many varices over the lower two-thirds of the thighs and all down the legs to the ankles, with prominent veins over each outer malleolus. There was pain on long standing and on walking. Both internal and external saphena veins were tied, and the varix over each outer malleolus was excised.	Seen February, 1902. There was much less pain—practically none. The veins were smaller, but were still visible.
49	29	M.	January, 1900.	Numerous varices in the right thigh and leg since 16 years of age. The internal saphena vein was varicose below the middle of the thigh, much so just below the knee; there were several large veins all round the leg. A large vein passed up the front of the thigh well to the outer side of the femoral vessels and appeared to enter the circumflex iliac vein. Pressure on the internal saphena vein did not control the varices; pressure on it and the vein to the outer side of the thigh did. He had lately had several attacks of slight thrombosis. Both veins were tied in the groin.	Seen December, 1901. Was very much better. He took active exercise—cycling, golf, shooting, and hunting—without pain. The internal saphena vein and tributaries had shrunk almost to normal size. The outer vein on the thigh was not materially altered; it appeared to have a deep communication just above the middle of the thigh, and through this it was filled by regurgitation.
50	38	F.	August, 1901.	Moderate varices of the right internal saphena vein in the thigh and leg. There were several dilated veins in the groin and the inner and upper part of the thigh. She complained of a great deal of pain, especially in the groin, which often unfitted her for her work.	Seen February, 1902. She had lost her pain, and was able to do hard work as a nurse without any discomfort—a thing that was impossible to her for years before. The veins had many of them disappeared, and all were much smaller.

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To sum up, I would say that Trendelenburg's operation is not only a simple and safe proceeding, but one that is very useful indeed in nearly all cases of varix. It may be relied upon to relieve the aching pain so often felt; it always stops further development of varices; it frequently leads to the shrinking, or even to the disappearance, of varices; and it exerts a strong influence in preventing thrombosis. Its good effects are most pronounced where regurgitation into superficial veins is best marked. Its superiority to excision of varices is greatest in cases of widely-distributed and numerous varicose veins, for it exerts its influence upon all the veins emptying into the saphena trunks. It is, therefore, useful in those widely-distributed, even general, varicosities of the lower limbs for which excision is inapplicable. To ensure success, the vein or veins must be obliterated quite at their junction with the deep veins. The fact that this operation has such a marked effect upon the course of varix is a strong proof of the important part that valvular defect plays in the history of cases of varicose veins.

LECTURE II.

On Obliterative Arteritis.

Mr. President and Gentlemen,—I have chosen "Obliterative Arteritis" as the subject of this lecture, partly because of the obscurity in which its origin and causes are wrapped, and partly because many of us are not so familiar with the main features of the disease as its frequency and gravity demand. It has barely found its way into our text-books, and it is still regarded as a very rare affection, and yet it cannot be so when from my own experience I can cite nine cases. The first important paper on the subject was written by Friedländer* in 1876, and was followed three years later by an article by Winiwater† giving a very careful description of the vessels in a case that had been under Billroth's care. Isolated cases were previously described by Jaesche‡ in 1865, and by Burow§ in 1867. But it is undoubtedly to Friedländer that the

* 'Centralblatt für die Medicinischen Wissenschaften,' 1876.

† 'Virchow's Archiv,' 1879.

‡ 'Archiv für Klinische Chirurgie, Langenbeck,' Band vi.

§ 'Virchow's Archiv,' Band xxxviii.

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credit belongs of recognising the condition, and of working out its pathology. Since his paper was published many examples of the disease have been described, but very little further light has been thrown upon it. In 1884 I* recorded a well-marked case affecting the vessels of the upper limb in a man, 19 years of age, and at the same meeting of the Clinical Society the late Dr. W. B. Hadden† recorded another case of the disease in the hand and forearm of a woman, aged 35 years. The following year Mr. W. J. Walsham‡ reported to the same Society a case of "acute obliterative arteritis" in the arm of a man, aged 52 years, but there is some room for doubt whether it belonged to this category or to that of acute spreading arterial thrombosis. In 1891 I§ read a paper on a case in which the disease caused symmetrical gangrene in the feet of a man, aged 43 years. In 1894 Saveran‖ recorded a case which began with an attack of phlebitis, after which there were persistent pain and lameness in the left foot; in a year the toes became gangrenous, and the leg was amputated. Saveran found endarteritis in the anterior tibial artery, and endophlebitis in the accompanying veins. In 1896 Mr. Hutchinson¶ described four typical cases, and called attention to the importance of examining the state of the artery in the thigh in all cases of gangrene of the foot. In 1897 a Russian surgeon, Wwedensky,** wrote of the disease as peculiar to Russia, and occurring in men between 19 and 60 years. In 1898 Mr. W. G. Spencer†† recorded a very remarkable case, in which the arteries in both legs and in one arm were simultaneously affected. Amputation through the middle of the left thigh was necessary, and Mr. Spencer gave an admirable account of the condition of the vessels in the amputated limb. About the same time Dr. W. A. Hollis‡‡ discussed the affection from its pathological side, and in 1900 Sternberg,§§ in a long article, described several cases of spontaneous gangrene in young people. The arteries of the limbs examined showed total obliteration of their lumen, with very vascular connective tissue internal to the elastic lamina; and

* 'Transactions of the Clinical Society,' vol. xvii.

† *Ibid.* ‡ *Ibid.*, vol. xix. § *Ibid.*, vol. xxiv.

‖ 'Bulletin de l'Académie de Médecine,' 1894, tome xxxi.

¶ 'Archives of Surgery,' vol. vii.

** 'Centralblatt für Chirurgie,' 1898, Band xxv, p. 937.

†† 'Transactions of the Clinical Society,' vol. xxxi.

‡‡ 'Journal of Anatomy and Physiology,' 1897–98, vol. xxxii, p. 153.

§§ 'Virchow's Archiv,' 1900, Band clxi, p. 199.

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Gibert* described a case of symmetrical gangrene of the feet from arteritis.

Before narrating any of the cases of this disease that I have seen I wish to make one or two preliminary observations. And, first of all, I would remind you of the paramount part that the inner coat takes in all active physiological and pathological processes in arteries. The prime office of an artery is to maintain a patent channel for the flow of blood, and this depends upon the integrity of the tunica intima. The physiological obliteration of obsolete vessels, such as the ductus arteriosus and the umbilical vessels, and the narrowing of vessels in stumps, are effected almost entirely by proliferation of cells of the intima—the middle and outer coats take no active share in the process. Similarly, the repair of a wounded artery or of a ligatured vessel is dependent upon the activity of the cells of the intima. If an artery is temporarily compressed or bruised, or its outer surface is cauterised, the changes produced are witnessed in the intima. If from any cause a thrombus forms in a vessel it is the inner tunic of the vessel which reacts to its presence, and if the blood circulating through a vessel is irritating or the tension high it is the same coat that suffers. The only primary change in the middle coat is one of simple degeneration—calcification—and we know nothing at all of any primary change originating in the tunica adventitia. The tunica intima is, in fact, the essential part of an artery; it is present throughout the entire system, and is equally important everywhere. The muscular coat is added as a vaso-motor mechanism, and the outer coat is an added strength to the artery, but both of these are subsidiary—there are vessels without the one or the other—but without the tunica intima there can be no vessel at all. Disease not only occurs in the inner coat of an artery more frequently than in either of the others—*e.g.*, atheroma—but there its effects are most serious. For when the inner coat is very gravely affected its function is interfered with and the lumen of the vessel is occluded, and this occlusion is brought about in one or both of two ways. In the first place, any hyperplasia of the endothelium or sub-endothelial tissue causes a bulging into the lumen of the vessel, the elastic lamina on the outer surface is a very resistant structure, and, as it were, shuts in the cells and soft tissues, and prevents their bulging out or passing into the muscular and fibrous tunics. The direction of growth is towards the centre of the vessel.

* 'Tr. Montpellier Médical,' 1900.

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But disease of the inner coat, especially when the endothelium is involved, very quickly leads to thrombosis, which, in small vessels, at once obliterates the lumen, and, except in rare cases, the clot soon becomes organised into vascular cellular tissue.

Of these two agents in the obliteration of arteries—new growth and thrombus—it must be borne in mind that while thrombosis may be, and often is, a very rapid process, the growth of the inner coat to such an extent as to fill the lumen of a vessel is always a slower process and slow in direct proportion to the size of the vessel. We must also bear in mind that a clot in an artery or a vein has a tendency to extend, and we often find it extend very rapidly; but the primary inflammatory growths of the intima appear to be much slower in their progress along an artery. And, lastly, another difference between these two occluding agents seems to be this —that a blood-clot may be absorbed and disappear, and the vessel which it has closed opened out again, but a like opening-out of a vessel once closed with sub-endothelial growth, so far as we know, never occurs. These considerations, in my opinion, make it very doubtful whether obliterative arteritis is a specific disease to be sharply differentiated; I would rather regard it as a pathological effect of many causes; and I would not therefore separate it from the syphilitic disease of small arteries described by Heubner and others.

The chief features of obliterative arteritis are these:—1. The disease originates in the subendothelial layers of the tunica intima of the smaller arteries. In its early stages it is marked by a small-cell infiltration which later organises into a loose and vascular connective tissue. 2. This growth narrows the lumen of the vessel and may entirely obliterate it; more often, however, thrombosis occurs, and the organisation of the clot completes the permanent occlusion of the artery. 3. The disease beginning in the smaller arteries tends to spread in a centripetal direction, and may reach even the largest arteries. The thrombosis it excites also often extends rapidly and far towards the heart, much faster and further than the changes in the vessel wall, and the clot may thus spread as far as the aorta. 4. The earliest effect of this diseased artery is pain, then follow other evidences of local ischæmia, and these may pass on to gangrene. These effects vary with the extent of the vascular obstruction and the efficiency of nature's means of compensation. 5. The disease may be very chronic, slowly progressing for years, or

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it may run a much more rapid course. Having reached a certain point arrest may occur, and the symptoms may gradually pass away as the unaffected vessels become more efficient substitutes for those that have been occluded. 6. The disease arrested for a time may afterwards recur in the vessels of the same limb or elsewhere, and it may attack more than one vascular area simultaneously. 7. The vascular changes are not always limited to the arteries, and endophlebitis may precede or accompany the endarteritis and the venous obstruction then modifies the effects produced on the tissues.

But I think we shall best appreciate the characters of the affection if I describe cases that I have seen, and I will take first of all one that I think is a fairly typical case; I will then narrate others to illustrate, as far as my experience permits me, the salient features which are met with, and I will conclude by drawing attention to some of the points not included in the summary just given.

Case 1.—A man, a carpenter by trade, was admitted into Middlesex Hospital under my care on July 2nd, 1895. He was then 56 years of age, a stout man, looking rather older than his years. He said that he had enjoyed good health up to October, 1894, when, he knew not why, the left internal saphena vein became swollen, inflamed, and painful from the ankle to the groin. For this he rested in bed, and the swelling gradually subsided. The following month (November) the left foot became red and "inflamed," and he was treated for some time for gout. The pain was great, and in spite of treatment continued all through the winter, and in March, 1895, the foot was noticed to be shrivelling and turning black. The gangrene gradually extended until it involved all the toes. Throughout his illness the pain had been very great, and he had been obliged to keep his bed. When admitted to hospital the toes of his left foot were black and shrivelled, blue-black gangrene extended on the foot as far as the mid-tarsal joint, and the skin of the rest of the foot up to the front of the ankle was dry and brownish. There was considerable œdema of the leg, and the skin was very sensitive; it was colder than the opposite leg and the skin was mottled and rather livid in hue. The left femoral, popliteal, and tibial arteries were pulseless and could be felt as firm cords. The arteries elsewhere in the body could be plainly felt pulsating; they were neither rigid, tortuous, nor uneven; they did not show any signs of atheroma or degeneration. There was no arcus senilis; the temporal pulse was not visible. The pulse rate was 136; it was rather uneven and compressible. The area of the cardiac dulness was diminished, the first sound was short, and the second sound was indistinct; there was no bruit. The man was pale, bald, with grey beard, with a good deal of soft subcutaneous fat; the abdomen was full and fat; the liver was not enlarged. The urine was acid, with a specific gravity of 1018, and it was free from sugar and albumen. The man was restless with a rather noisy delirium, but was evidently in great pain. The temperature varied between 100° and 101° F. There was no history or evidence of syphilis. On the lower part of the right leg was a pigmented scar of an ulcer. I amputated the limb at the knee-joint. There was no

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bleeding from the popliteal artery when the tourniquet was removed, and the end of the vessel could be plainly seen securely plugged by soft solid vascular tissue. There was slight oozing of blood from vessels in the sheath of the artery, showing a very vascular condition of that sheath; only five ligatures were needed for the whole stump. Immediately after the operation the patient lost his delirium and his pain, he became quite quiet, and his general health rapidly improved. The stump healed well and the temperature fell to normal on July 9th, and on the 24th he went to a convalescent home at Swanley. (Owing to an accident the amputated member was not dissected.) For two years this man continued well and got about freely. But during the winter of 1897-98 he came to me at the hospital several times complaining of severe pain in the right foot and leg and a sense of coldness and numbness—these pains were much worse when the weather was very cold, or if the part was not warmly covered up. I found the foot and leg as high as the knee cold and mottled in colour, with very sluggish circulation, and I could not detect any pulsation in either the tibial or the popliteal artery. I advised him to rest the limb and to keep it very warm. It was evident that the disease had now affected the arteries of the right foot and leg. On his last visit, in the early summer of 1898, he was rather better. But he was suddenly readmitted to the hospital on October 21st, 1898, in a very grave condition. He was incoherent, unable to answer questions, restless, and complained of pain in his foot and leg. His tongue was thickly coated with a white fur, and he seemed to be very thirsty. His friends stated that his foot had become cold and blue on the 19th, and that after that he had become rapidly more ill. I found the right foot and lower third of the leg very cold, mottled blue and white, slightly swelled, and the circulation, although nowhere arrested, was yet very sluggish. There was no pulse in the tibial, popliteal or femoral arteries; they were all, when felt, firm cords. The arteries of the arms, on the other hand, were soft and not tortuous. The pulse was rapid, regular, and compressible. The urine was pale, of specific gravity 1005, acid, free from sugar, with a slight cloud of albumen on boiling. The temperature ranged between 99° and 100° F. I kept the limb as warm as I could with thick cotton wrappings, outside which were placed hot bottles, and tried to relieve his pain with morphia. But he got worse. On the 24th I could not demonstrate any circulation in the foot; in the lower third of the leg it was very languid, but the upper half of the leg was warmer and the circulation was fairly active. The superficial veins were not thrombosed. His temperature was higher (101°), he was less conscious, and sometimes passed his urine in the bed. Remembering his rapid improvement after the former amputation, I decided to remove the limb at the knee, and did it on the 24th. He slept well after the operation, and woke up quieter and more rational. The next afternoon, however, he became more restless; his temperature rose; on the 26th it reached 103·8°, his pulse was counted at 156, and just before midnight on the 26th he died.

On dissecting the leg we found the popliteal artery blocked by a firm organising clot. The posterior tibial artery was quite small, occluded in its entire length by a soft colourless clot; the wall of the vessel was considerably thickened, especially at the posterior part of its circumference. The anterior tibial artery was small, with thickened walls, but had still a narrow lumen. The arteries were very firmly matted to their companion veins, and were separated only with great difficulty. The *post-mortem* examination was made by Dr. A. F. Voelcker, who recorded that the heart was dilated, flabby, with *ante-mortem* clots in both ventricles and

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in the right auricular appendix. The aorta in the thorax showed very trifling disease, and there was no atheromatous ulceration. The carotid and subclavian arteries and the jugular, innominate, and superior caval veins were normal. The femoral and iliac vessels were blocked with clot, the lumen of the vessels was only a little diminished, and, except for some calcareous patches, the walls did not show any marked change. The clot extended up the abdominal aorta as high as the origin of the renal branches, and here ended in a conical tip. The left kidney showed one infarct; the right had many.

This case is fairly typical of the affection called obliterative arteritis. I would call attention specially to the insidious onset, slow progress, the severe pain coming on as the first symptom and lasting throughout—pain that was worse in cold weather, or when from any cause the affected part was exposed to cooling. Then the signs of local ischæmia, with loss of pulse in the arteries and their conversion into solid cords, passing at length into gangrene of the most distant sections of the member—all this, associated with a healthy condition of the arteries generally, as shown by their softness, smoothness, and absence of convolution and loco-motor pulse at the elbow and temple, for instance. The symptoms may vary in intensity a little from time to time, but there is never the marked periodicity or intermission of symptoms that characterise Raynaud's disease. The obstruction to the circulation, whatever it is, is organic and permanent from the first, and capable of relief only by the agency of anastomosing and alternative arteries. The essence of Raynaud's disease is vascular spasm, variable and temporary, and only causing permanent changes—gangrene—when prolonged spasm is followed by thrombosis. Hæmoglobinuria, which often occurs in Raynaud's disease, is never present in obliterative arteritis. The case which I have quoted is instructive, too, in the recurrence of the malady in another limb, and this event is so important that I will briefly quote another case in which I have seen it.

Case 2.—A man, aged 60 years, a carpenter, was admitted into Middlesex Hospital under my care on May 12th, 1897. There was no family history of importance. His general health had always been good; he had been a temperate man, and had not had syphilis, gout, or tubercle. He told us that he was subject to chilblains, especially when young, and that he had injured his right leg and foot on two occasions: once, 28 years before we saw him, when the lower part of the leg was "crushed," and it took six months to heal up. Eighteen years later the right great toe was contused; the nail separated, and had never grown again. He had been laid up for three and a half years when I first saw him. The trouble began, he said, with a sore formed on the left heel, which became black (gangrenous); later the little toe, then the second toe, and

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afterwards the third toe became gangrenous; part of the second toe had separated spontaneously. Four months before admission he noticed a black spot on the great toe. Two months later the skin over the dorsum of the foot became blue, and this spread to the leg. All this time he had suffered from dull, aching pain with sharp twinges in a part before it became black. He was a dark-haired, thin, emaciated man. He was depressed, haggard-looking, and complained of great pain and inability to sleep. The left foot and ankle were blue and cold, and on the outer side this extended as high as the middle of the leg. The three inner toes were mummified; there was a large patch of dry gangrene on the dorsum of the foot; the sole of the foot was purple and cold, but not actually gangrenous. No pulse was felt in either tibial artery; there was slight pulsation in the popliteal artery. There was some matting of tissues over the dorsum of the right foot and the lower fourth of the leg; the skin was thin and atrophied; there was no loss of sensation or of pulsation in the arteries. The heart was healthy; the temporal artery was not visible or tortuous; there was no locomotion or tortuosity of the brachial artery at the elbow. The urine had a specific gravity of 1017; it was free from sugar, and it contained excess of phosphates and a faint trace of albumen. The temperature ranged from 100° to 101° F. I amputated the left leg at the knee-joint; only three arteries were tied—the popliteal, a large branch coming off from it and the internal sural artery; the popliteal artery was thickened with a narrow lumen and would not twist. The arteries in the amputated leg were found to be all greatly thickened; the dorsal artery of the foot and the external plantar artery were occluded, the internal plantar was still patent. The veins in the sole were thrombosed, those in the leg were pervious. The man made a slow convalescence, part of the outer flap sloughed, and the bone had to be shortened. But he eventually left the hospital for the convalescent home in August, 1897, free from pain, sleeping well, cheerful, and quite a different man from when he came in.

He remained well till March, 1899, when he found that the inner side of his great toe was tender and he began to suffer pain in the foot. At the end of September a black patch appeared at the root of the great toe, and the gangrene spread to the other toes and the dorsum of the foot. The pain meantime increased to great severity, described as aching in character, with sharp twinges or paroxysms. He was again admitted into the hospital on November 29th, 1899, having been ill nine months. The left stump was sound and well; it was very difficult to detect any pulsation in the femoral artery on that side. The right foot was swollen, cold, mottled in colour, and the toes were gangrenous. He complained of intense pain in the foot, and was evidently in great suffering. There was no pulse to be felt in any artery below the femoral. The radial artery was not hardened or tortuous, and there was no evidence of disease in other arteries. The urine was of specific gravity 1020, free from sugar, and contained a trace of albumen. On December 2nd I amputated through the condyles of the femur by Lister's method. The popliteal and two or three small arteries were tied: the wall of the popliteal artery was much thickened. Convalescence was again slow, but he went to Clacton with the stump healed on January 11th, and returned on February 6th, having gained 13½ lbs. in the four weeks. I have seen this man occasionally since. The left stump has given him no trouble, but on his last visit to me the right thigh was very painful: it was cold, rather blotchy and blue, and I could not feel any pulse in the femoral artery. I advised him to keep it very warm.

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The liability for the disease to attack both lower limbs was illustrated by a case that I reported to the Clinical Society, and which will be found recorded in vol. xxiv of that Society's Transactions.

CASE 3.—The patient was a man, aged 43 years, a draper's traveller. While an inmate of an asylum for acute mania supervening upon an attack of influenza he was seized with severe pain in his feet and legs. They were noticed to become cold and blue, and in a few days gangrenous. When admitted to the Middlesex Hospital there was gangrene on each side as high as the bases of the malleoli, with mummification of the toes and œdema of the legs. Pulsation could not be felt in the politeal or tibial arteries, the heart sounds were normal, the pulse was 88, and the brachial arteries were not obviously diseased. The urine was acid, free from albumen and sugar. I amputated the legs at the knee-joints, and the patient made a good recovery. I have heard nothing of him since. The popliteal arteries, where divided, were filled with organising clot, and the wall of the vein was thickened. The arteries and some of the veins in the leg showed marked thickening of the interior coat with, in places, occlusion of the lumen by coagulum; there was no calcareous degeneration.

These three cases will suffice to illustrate the tendency of the disease to attack the vessels of more than one limb, either concurrently or successively. Mr. Spencer has recorded a very interesting case in which three limbs, the right arm and both legs, were simultaneously affected.

Let me now relate some cases from which I think we may learn something of the etiology of this affection. The first of them is that of a woman who developed symptoms of erythromelalgia after an attack of influenza, and then shortly afterwards showed signs of local ischæmia in the same limb caused by arterial obliteration.

CASE 4.—The patient was a woman, aged 65 years, seen in consultation with Dr. Ker and Dr. Aldren Turner. I am indebted to Dr. Turner for the notes of the earlier history of this patient. This patient had enjoyed good health until the early part of 1898, when she had an attack of influenza. She made a good recovery, and remained well through the summer. In October, 1898, she experienced pain in her right foot without any loss of power. This continued more or less all through that winter and the following spring, and in August, 1899, she consulted Dr. Aldren Turner. He found marked tenderness over most of the nerves of the upper and lower limbs; she complained of pain in the right foot, and over the toes there was marked superficial pain, with redness and increased heat. She could scarcely bear to place the foot on the ground, so great was the pain produced. There was no paralysis. She was thin, and all the tissues were flabby. Dr. Turner ordered galvanism. When next seen in September there was marked improvement in the foot —apparently as the result of the galvanism. General massage and the recumbent position were then ordered, and bromide of potassium,

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salicylate of soda, and phenacetin were prescribed. On October 6th Dr. Turner received a letter from Dr. Ker, stating that there had not been any improvement lately, and the treatment seemed to have lost its beneficial effect. But very soon after that the patient appeared to get quite well, she went about freely, walking was easy to her, and she went on a visit into the country. On January 14th, 1900, Dr. Turner visited her in the country, and learnt that she had continued well until the onset of very cold frosty weather about 14 days before Christmas, when she had great pain and a burning sensation in the right foot, followed shortly after by signs of local asphyxia in the toes—chiefly the second toe. At his visit he found all the toes of the right foot blue, there was a large blue patch over the right heel, and another over the dorsum of the foot. The foot felt moderately cold to his hand, but subjectively was said to be very cold. The toes were very tender; there was no anæsthesia. The pain in the foot was intense at times, chiefly at night. There was no pulse in the posterior tibial artery; the radial pulse was of good volume, and there was no sign of marked atheroma; and no affection of the nose or ears was present. The patient complained of slight pains in the left toes, where there was some doubtful redness, and also of slight numbness in the fingers. The pain continued very severe, the condition of the foot became worse, and I was asked to see the patient on January 24th. There was then gangrene of the toes and dorsum of the foot and the heel. I could not detect pulsation in any of the arteries of the right lower limb below the middle of the thigh; the arteries could be felt as firm cords. Pulsation could be plainly felt in all the arteries of the left thigh and leg. The urine contained lithates, but was free from sugar and albumen. The radial, brachial, and temporal arteries did not show atheromatous change. The patient was thin, much worn by her suffering ,and her appetite was failing. She got very little sleep. The heart and lungs and the liver were healthy. The right kidney was movable. There was no evidence of syphilis or of alcoholism. We advised amputation, and next day I removed the limb just above the knee-joint. The popliteal artery was a solid cord and did not require a ligature. She made a good recovery from the operation, the stump healed well, and the pain was at once greatly relieved and the patient's general condition correspondingly improved. I was, however, asked to see this patient in March—two months after the amputation—and I found that she was then suffering from severe pain in the thigh. This was swollen quite considerably, and the skin was marked with blotches of blue and red; the surface was cold. The pain in the thigh was described as very severe. I could not feel any pulse in the femoral or external iliac arteries. The arteries on the left side pulsated normally. I advised that the limb should be kept raised, very warmly swathed, and gently massaged twice a day. At the same time I gave a serious prognosis, as I feared that the disease might extend to some visceral artery. I did not see the patient again, but Dr. Ker has informed me that the pain, swelling, and discoloration of the thigh disappeared, and the patient seemed to get "quite well," so far as the arterial disease was concerned. She died in 1901 from some chest affection—not from anything connected with the old trouble. She was not then under Dr. Ker's care, and I have not been able to obtain more precise information. The arteries in the amputated limb showed the usual changes. The popliteal, tibial, and peroneal vessels, as far as they could be traced, were converted into solid cords with greatly thickened walls. The tunica intima was much thickened, and the narrowed lumen was occluded with organising thrombus.

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This case is specially interesting because of the history of the onset of the affection—unique in my experience and reading. When Dr. Aldren Turner first saw this patient she presented the symptoms of erythromelalgia—that is to say, neuralgic pains, with signs of dilatation of the arteries and increased vascularity of the tissues. There was local heat, and the pain was greatly increased by putting the foot to the ground and by warmth. This was the state of things in the summer of 1899, and these symptoms passed off and the patient appeared to be quite well in the autumn. But with the onset of wintry weather the picture was strangely altered: the local heat and flushing were changed into coldness and sluggish circulation, which gradually culminated in gangrene. Erythromelalgia is a rare affection, and but little is known about it. In his excellent article in Clifford Allbutt's 'System of Medicine' Sir Thomas Barlow sums up the evidence for and against the various views of its pathology that have been advanced, and shows that the balance of evidence is in favour of the view of its consisting in a dilatation of the arteries caused by paresis or inhibition of the vaso-motor nerves. Whether this be a primary or a sequential phenomenon the dilated arteries are the most important feature of the disease, and Sir Thomas Barlow very pertinently remarks that the arterial dilatation might excite changes in the intima and lead to thickening of the arteries and narrowing of their tissues; he refers to Thoma's experiments to show that increased arterial tension, if kept up, leads to proliferation of the intima. The case I have related shows us this very sequence. It is also of interest as an illustration of the tendency of the disease to spread upwards, even after amputation well above the gangrenous area, and of the smaller gravity of the disease in the thigh than in the foot and the leg.

The next case illustrates another cause of the same condition.

Case 5.—The patient was a woman, aged 45 years, seen in consultation with Dr. S. Blake Jolly. For many years she had suffered from very severe menorrhagia which had kept her in a state of extreme anæmia. For years she had taken alcohol in excess, chiefly in the form of spirit. She was constantly needing medical attention, either for the hæmorrhage, or the anæmia, or the effects of special alcoholic excess. Ten days before I saw her Dr. Jolly was called in, and found her in great pain in her left foot with paroxysms of very severe agony; the foot was colder than the other, pale, mottled in colour, with sluggish circulation. The severe pain continued in spite of the application of local heat, and injections of morphia had to be given. I saw the patient on March 6th, 1900; she was very stout and flabby, with a very fat abdomen; her skin was white

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and her mucous membranes were very pale, and she looked very unhealthy. The toes of the left foot were gangrenous, blue-red in colour, not altered in size, and the gangrene extended for about an inch on to the dorsum of the foot and a little further on to the sole. The rest of the foot was cold, mottled blue, and white in colour, but the skin retained its sensitiveness as far down as the edge of the gangrene. I could not feel a pulse in the popliteal artery or either of the tibial arteries; there was pulsation in the femoral artery and in the tibial arteries of the right side. There was no evidence of general atheroma of the arteries. The brachial artery was not tortuous or hard. There was no valvular disease of the heart. The urine was free from sugar, but it contained a very slight trace of albumen. This patient was in very severe pain in the foot and leg. I recommended amputation, and next day removed the limb above the knee-joint. This patient made an excellent recovery. I heard of her quite recently from Dr. Jolly, and he told me that her stump had remained well, so had her left foot and leg, and that she had continued the abstinence from alcohol which we enforced during the healing of the wound, and with very great benefit to herself. I think the *causa causans* in this case was the alcoholism—the irritating effect of the alcohol circulating in her blood. The case illustrates also what we so often see—the great benefit which alcoholic patients derive from breaking off the habit and excluding alcohol altogether from their dietary, even when organic change, such as obliterative arteritis, has taken place. If this patient will continue her abstinence from alcohol I am very hopeful that she will have no return of the disease. She has been free from it for two years already.

In the following case the condition probably resulted from syphilis. This patient was in the Middlesex Hospital, under the care of Dr. W. Pasteur, and I am indebted to him for an opportunity of examining the man and for permission to use the notes of the case.

CASE 6.—A man, aged 48 years, a gardener, was admitted into Middlesex Hospital on January 3rd, 1898. He had had good health up to the time of this illness, and we failed to elicit from him any history of syphilis or of injury. He said that for three and a half years he had suffered from redness and a burning and stinging pain in the left great toe at night, so severe as to prevent sleep. This pain sometimes came on during the day. Similar symptoms came on in the right foot about six weeks after he noticed them in the left. The feet were at times quite white, at others blue, and he noticed then that when he pressed the colour out with his finger it returned very slowly. The great toes suffered most, the other toes to a less extent. He had not noticed any numbness or loss of sensation. Six months before he came under observation he observed mottled redness of the soles of the feet, and a month before admission he saw a blister on the tip of the right great toe; this gradually extended under the nail, and beneath it a black eschar was seen. This change was accompanied by most excruciating pain. He was a man of spare build and nervous habit; his pulse was 72, weak and compressible; the heart sounds were healthy. The urine was acid, 1020, and there was no albumen or sugar. The temperature ranged between 97·6° and 99° F. The skin of each foot, especially on the sole, was much

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mottled and generally reddened; on the end of the right great toe was a patch of black gangrenous skin. He suffered great pain, and even hypodermic injections of morphia failed to give him more than two hours' sleep at a time. There was no pulse in either posterior tibial artery or in the left popliteal and femoral; that in the right femoral artery was quite distinct. The gangrenous patch on the toe extended a little, the slough slowly separated, and the ulcerated surface still more slowly healed. He was sent to Clacton on February 27th in good general health, nearly free from pain, but still showing blotchy discoloration and sluggish circulation in both feet. The man returned to hospital on April 13th, 1898, on account of increasing pain in the left foot. The right femoral pulse was only just perceptible, the left could not be felt, and the artery was represented by a hard cord in Scarpa's triangle. No arterial pulse was felt in either limb below the femoral artery. The toes of the left foot were of dark blue-black colour and "somewhat swollen." There was very distinct "marbling" of the foot, with purple and red and paler areas. The lividity of the toes increased, blisters formed and burst, and exposed foul, discharging surfaces, and slowly in a month or so the toes were quite gangrenous. All this time the patient suffered very severe pain. The urine was sometimes quite normal; more often it contained just a trace of albumen. Nothing was detected abnormal in the arteries of the upper limbs. On May 27th the urine was noticed to be of a dark orange colour, of specific gravity 1020, no albumen or sugar. This colour persisted up to the man's death. Albumen was sometimes present in small quantity. It did not give the reaction for blood-pigment, bile, or indican. On the 31st the gangrene of the foot was found to be extending towards the heel. He was frequently sick, and was unable to retain any food. On June 3rd the vomiting, with much retching, returned, and persisted until the man died from exhaustion on the 8th. On the 7th a tumour was discovered occupying the greater part of the lower half of the abdomen. Its outline was indistinct, and it was dull on percussion. Above it percussion revealed a distended stomach. This "tumour" was not present three days before; it was uninfluenced by emptying the bladder by a catheter.

At the *post-mortem* examination a gumma was found in the liver as well as definite syphilitic infiltration; the vessels of the liver were not thickened. A gumma was also found in the spleen. The lower 21 inches of the ileum were firmly adherent to the anterior abdominal wall and to each other. This portion of the intestine was purple-black in colour, without any peritoneal lustre, and its coats were greatly thickened by an infiltration of coagulated blood, so that its lumen was considerably diminished. The intestines above this was greatly distended. The colon was filled with feculent material of a dark coffee-ground colour. The mesentery corresponding to the affected part of the bowel was much thickened from extensive hæmorrhage into it. The superior mesenteric vein was distended with a thrombus quite up to the commencement of the portal vein. The superior mesenteric artery was markedly thickened, and its lumen was almost obliterated in parts. A thrombus extended from the upper end of the left external iliac artery to the popliteal artery, and on the right side a clot extended from the upper end of the external iliac artery down into the tibial arteries two inches below the bifurcation of the popliteal. The heart was healthy, and the abdominal aorta was healthy except for small areas of atheroma around the mouths of branches. A microscopical examination of the vessels showed distinct thickening of the intima of the superior mesenteric

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artery with a loose connective tissue; in the arteria dorsalis pedis there were similar changes, except that the endothelium, though irregularly thickened, had a dense structure as compared with the loose one seen in the mesenteric artery.

In this case the cause of the disease was probably syphilis. There were old endarteritis of the vessels in the feet, more recent extensive thrombosis of the arteries above, and in addition arteritis and (possibly) phlebitis of the superior mesenteric vessels with thrombosis leading to great hæmorrhage into the walls of the intestine with corresponding loss of function.

I have seen two cases of this disease in the upper limb, one of which is very fully recorded in vol. xvii of the 'Transactions of the Clinical Society.'

Case 7.—This case occurred in a young brickmaker. The painful illness extended over about eight months, and the main arteries of the limb were gradually obliterated up as high as the upper end of the brachial artery. There was gangrene of the tips of two digits. While the disease was active the pain was most intense.

Case 8.—This case occurred in the right arm of a woman between 30 and 40 years of age, whom I saw on one occasion only as she was passing through London. After an attack of influenza she began to suffer with severe pain in the right hand and forearm, made worse by using it and by exposure. The pain was attributed to neuritis; remedies had no effect upon it. When I saw her she was much better, and was returning home after a visit to Bournemouth. I found the radial, ulnar, and brachial arteries as high as the middle of the arm converted into firm pulseless cords; the forearm was a little wasted, tender, bluish-red in colour, not so warm as its fellow, and painful if allowed to get cold. The other arteries of the body and of the heart were healthy, so was the urine.

Case 9.—A man, aged 48 years, was seen by me once only in 1896. He had lived a healthy, active life, and there was no history of rheumatism, gout, or syphilis. In the autumn of 1890, when out shooting, he suddenly felt his right leg become cold, weak, and painful; this was not due to any injury, strain, or exposure to cold. He lay up for many weeks at the suggestion of his medical man, who told him that there was no pulse in his posterior tibial artery. He remained well until September, 1891, and then, again when out shooting, he was seized with the same kind of pain in his left foot. This attack was more serious than the former one; the pain was very severe, and the second toe became almost gangrenous. Among others he was seen by M. Charcot, who prescribed prolonged rest in the recumbent position. He rested the leg for six months. After that he had two attacks of phlebitis—one in each thigh. When I saw him in 1896 he could walk with comfort for 100 yards, but then a painful sense of weakness in the legs came over him. When standing he was always desirous of sitting or lying down. Each leg was slightly œdematous, and the circulation in the feet and lower part of the leg was distinctly sluggish. On the right side there was no pulse in either of the tibial arteries; on the left side there was no pulse in

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the anterior tibial, and only a slight one in the posterior tibial vessel. Both femoral arteries were normal, and so were the arteries of the head and neck. There was no disease of the heart, or any sign of aneurysm; the urine was 1018, acid, and free from albumen and sugar. The liver and spleen seemed to be healthy. I recommended this patient to use his legs a little more and to have them carefully massaged. I have not seen him since. The history of this case is certainly peculiar. The sudden onset of the symptoms is suggestive of embolism, but there was no obvious source of such. The recurrence of the attack under exactly similar circumstances is striking, and yet I cannot see what clue it gives us. In other respects the case resembles endarteritis, and in each leg the obliterated vessel could be clearly demonstrated.

These cases illustrate several points in the history of this affection, in addition to those which I mentioned in my preliminary summary. I will refer to a few of them. The *extreme chronicity* of the process is well exemplified by Cases 2 and 6, where it had lasted for three and a half years before I saw the patients. Cases 3, 4, and 5 are, on the other hand, examples of a more acute form of the malady, and intermediate grades are furnished by Cases 1, 7, and 8. The *pain* which is experienced by these patients is generally very severe. I have never seen stronger evidence of extreme pain in anyone than I witnessed in the young man whom I saw in 1884 (Case 7). He often writhed on the floor in agony that even large doses of morphia had little effect in controlling. Pain is the earliest symptom noticed; it has been described as "rheumatic," also as pain in the bones; it is referred to the deep structures of the part, and is aching in character, and the intensity of this ache may culminate in an agony. It is often worse at night, is generally made worse by use of the part, and is always aggravated by exposure. The *ischæmia* varies in degree. As a rule there is not much alteration in colour until gangrene is threatened; what there is is blotchy in distribution and dull red. It is specially noteworthy that it is not accompanied with anæsthesia. There may be slight wasting of the part or swelling from œdema—this generally due to venous obstruction. The *gangrene* may be a slow mummification or a more rapid sphacelation; in some cases the parts preserve their consistence and proportions quite remarkably, being neither "dry" nor "moist." This variation, no doubt, depends upon the degree to which venous obstruction is associated with the arterial obstruction, if, indeed, it is present at all. The case which Dr. Pasteur has allowed me to use (Case 6) showed the extreme degree of venous thrombosis in the mesentery with extensive

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hæmorrhage into the bowels and between the layers of the mesentery. Another feature of the gangrene often noticed is its patchy distribution—in some cases, no doubt, it is total up to a given level (Case 3), but in others (*e.g.*, Cases 1 and 2) separate areas of the foot necrose. This is easily explained by the irregular extension along the arteries of the obstructive disease. *Fever* is usually present when the disease is active (*see* Cases 1, 2, and 7), and other constitutional effects may be witnessed, such as wasting, loss of appetite, and delirium. The state of the *other arteries* of the body is naturally laid great stress upon in diagnosis. Widespread atheroma or calcification affords an obvious explanation of interference with the circulation in distal parts; but, of course, this disease may occur in patients who manifest some degree of atheroma, and an instance of this is afforded in Case 1.

Sex.—The disease is certainly more common in men than in women—in my cases in the proportion of seven to two.

Age.—My youngest patient was 19 years of age and my oldest was 60 years of age. It seems to be a disease of adult life, not occurring in infancy or in old age. Dr. Bryant and Dr. W. Hale White have recorded a remarkable case in the fifty-fifth volume of the 'Guy's Hospital Reports' of an infant, 6 months old, who had gangrene of the toes and distal half of the foot. The child was greatly emaciated and died. There was widespread arterial disease, the inner coat was greatly thickened, and there was calcification of the deep layers of the intima and of the media, so that the artery cracked when bent and felt gritty. I hesitate to include this case in the same category as those which we are considering. It seems to stand out as almost unique—possibly it is of the same nature as one recorded by Durante.*

Etiology.—Various views of the causes of this form of endarteritis have been advanced. Friedländer attributed it to the influence of injury and external cold. Wwedensky believes that there is a neuritis associated with, and possibly antecedent to, the arteritis. Sternberg attributed it to the effect of various injuries upon a weak and depressed vascular system. Hollis suggests that by the repeated impact of foreign particles against the sides of a small artery a state of "irritation" or "molecular unrest" is established within certain endothelial cells. This unrest manifests itself physiologically by

* 'British Medical Journal,' Epitome, 1899, vol. i, p. 542.

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rousing the little central mass of nuclear protoplasm of the cell from a dormant condition to activity. We really know so little of the ultimate causes and processes of disease that it is difficult to accept or to reject such theories as these. Of all known causes syphilis is perhaps the most certain; influenza, alcoholism, erythromelalgia, cold, contusions, and previous thrombosis or phlebitis may be regarded as more or less important factors in originating the disease. Diabetes sometimes causes gangrene by exciting this disease.

Treatment.—There is little to say on the subject of treatment. In the earlier and less advanced stages of the malady prolonged rest of the affected part is certainly called for. Local warmth should be carefully supplied. Anodynes, often in large quantities, may be necessary for the relief of pain. Massage carefully carried out has also an important influence in furthering the opening out of collateral vessels, in promoting absorption through the lymphatics, and in freeing the muscles and other tissues of their waste products. In the acute stage the parts are too tender to admit of it; in all early cases it must be most cautiously employed; where the disease seems to be arrested, and we have to deal with its effects only, it may be used a little more vigorously. Small mummified areas may be left to separate naturally, especially in the hand. For more extensive gangrene, which is always, I believe, of the "mixed" form, amputation is necessary; the relief to pain that the operation affords always reconciles the patient to the mutilation. The operation should be done high above the gangrenous spot, and at a place and by a method that throws least strain upon the vessels of the part. Thus, amputation through the lower part of the thigh is to be preferred to disarticulation at the knee, and musculo-cutaneous flaps are to be chosen in preference to skin flaps.

LECTURE III.

Mr. President and Gentlemen,—The very interesting problems connected with the occurrence of thrombosis, and the great practical importance of the affection are, I hope, a sufficient justification for my devoting this lecture to that subject. Ever since the science of physiology has existed, the coagulation of the blood has been

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a phenomenon of absorbing interest to physiologists, and although many keen observers and profound thinkers have devoted much patient labour to its study, we have to confess that it remains to-day in great measure an unsolved mystery. But if the coagulation of the blood is one of the great elemental problems to the physiologist, thrombosis, or the coagulation of the blood in living blood-vessels, is a theme of equal interest and importance to the pathologist and to the practitioner of medicine and surgery. To its beneficent agency we owe the very existence of our race. The great function of reproduction seems to be specially dependent upon the benign influence of thrombosis in sealing the tiny vessels in the ovary when an ovum bursts from the Graafian follicle, in closing the ruptured vessels of the torn hymen, and in plugging the much larger uterine sinuses which are opened by the separation of the placenta. The whole art of surgery may, without any exaggeration, be said to have been possible only because blood may coagulate within living vessels, and the debt of surgery is perhaps only fully realised when one stands almost impotent beside the ceaseless dripping from a wound in a hæmophilic patient. For most of us, I fear, the word "thrombosis" conjures up before our minds not these benign effects, but some of those disastrous results which we so often see following upon the untimely clotting of blood within the vessels. We think of pains and long-drawn-out disabilities, of extended periods of rest and idleness, perhaps of permanent lameness or paralysis, or even of sudden death, or the *ante-mortem* agony, all of them so often resulting from thrombosis. Indeed, it plays almost as prominent a part in disease and death as in the preservation of health and life; perhaps we hardly realise how large a part it plays, and it is not uninstructive to use some spare moment by trying to count up the many and different forms in which we meet with thrombosis, and how enormous an advance in practical medicine it will be when thrombosis will be known only as a beneficent phenomenon, and when we are able to prevent its occurrence as a baneful process. As a very minor illustration of the importance of thrombosis, and of the large influence it may have in marring a life, I may mention a case I saw last year of a man, 49 years of age. He told me that in 1876 he was attacked with typhoid fever, and during his convalescence he suffered from thrombosis of first one femoral vein and then the other; for this he was laid aside for 18 months. Ever since that time he has been

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liable to thrombosis from slight injuries—and, indeed, it has often occurred without obvious exciting cause—and out of 25 years he has spent no less than six years, two months, and one week invalided by these successive clottings! When I saw him last July he had plugging of the left median basilic and the lowest inch of the basilic vein, which had quickly followed upon his being rather roughly seized by the left elbow. I feel sure that all of you can recall case after case where blood-clotting has proved almost as great a bar to a happy, useful, vigorous life as in this case. I forbear to speak of those many tragedies that we witness as the result of the sudden detachment of one of these clots.

It is no wonder, then, that the literature of thrombosis is very extensive, and that many of the greatest pathologists—John Hunter, Cruveilhier, Virchow, Paget, Lister, Cohnheim, and a whole host of others—have made it a special study, and have endeavoured to fathom the deep mystery that covers the origin and nature of intravascular coagulation. I cannot attempt to sketch the history of the theory of thrombosis, how it has gradually been freed from error and misconception, and amplified by new facts. I propose only to touch upon a few points, and to mention one or two of the less commonly seen forms of thrombosis that have happened to come under my observation.

The first appearance observed in circulating blood about to clot is the presence of minute protoplasmic masses called "platelets," which, becoming increasingly viscous, cohere into tiny irregular masses. These platelets are probably not constituents of normal circulating blood, but are derived from the red corpuscles, possibly from their disintegration. Their increasing viscosity is probably dependent upon some unknown condition of the blood plasma. Around these minute masses of agglutinated platelets fibrin is deposited, and encloses corpuscles in its meshes if the blood-stream is not so turbulent as to "whip" them out of the forming fibrin. This fibrin is deposited from the blood in such a way as to impart to the clot a definite structure. But the question that interests us most is, Why do these clots form? What is the condition antecedent to the formation and agglutination of the platelets? Is it to be sought in the blood or in the blood-vessel? These are questions as old as the scientific study of pathology, and a full and completely satisfactory answer to them is not yet forthcoming. John Hunter regarded a plastic inflammation of the vessel wall as the primary

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and essential step in the whole process, and Cruveilhier advocated the same view. Virchow, in his great work on thrombosis and embolism, laid stress on the opposing view, and regarded the coagulation as a blood change effected by some alteration in the blood itself, independent of changes in the vessel-wall, which, however, were often set up by the presence of a blood-clot. More lately, and as the outcome of the study of micro-organisms, and the part they play in so many forms of disease, it has been shown that in many thrombosed vessels bacteria are present both in the vessel-wall and in the clot, and it is thought that these organisms may be the primary cause of the coagulation. Opinions differ, however, as to whether the micro-organisms produce the clotting by acting directly upon the blood-cells, or whether, by their presence in the vessel-wall, they set up changes which unfit them for their special function of maintaining an open channel for the blood, and thus lead to blood coagulation. It is evident that if a blood change is the chief cause of intravascular coagulation, there must also be some local condition that determines the thrombosis at a particular part of the system. This is a problem of exactly the same kind as that presented by other local manifestations of general infections, such, for example, as acute infective osteo-myelitis, tuberculous and syphilitic osteitis. In each case there is a general infection and a localising influence.

As regards the blood itself, we must abandon the idea that a condition of hyperinosis—an excess of fibrin—ever plays any part in the causation of thrombosis. The proportion of fibrin present in the circulating fluid no doubt varies within somewhat wide limits, but the greater or less tendency to thrombosis never depends upon the more or less of fibrin present, but upon some agent that excites the separation of the fibrin from the plasma. The blood changes that might bring about thrombosis we may suppose to be one or more of the following:—(1) A change in the corpuscles leading to their disintegration; (2) an increase in the viscosity of the platelets; (3) an increase in the fibrin ferment; and (4) an increased liberation of fibrin ferment. It is possible that various micro-organisms or their toxins may influence in some way or other either the formation of platelets from red corpuscles or the production of fibrin ferment in the blood. It is also possible that certain products of inflammation absorbed into the general blood-stream may have the same effect. Bearing upon this question is the

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well-known fact of the tendency of a clot once formed to extend—in the direction of the circulation, it is said. This growth of a clot is sometimes very rapid and very extensive, and one explanation of it is that there escapes from the clot first formed some element capable of exciting coagulation in the adjacent blood, fibrin ferment possibly. If this is so it affords support to the view that a blood-change may be both the primary step in thrombosis, and the direct cause of the coagulation. Conversely, it has been shown that a state of the blood may be induced in which coagulation cannot occur, and it is thought that an exhaustion of fibrin ferment may actually be the nature of this change. The late Sir William Roberts suggested that gouty thrombosis might be due to the presence in the circulating blood of minute particles of uric acid, which he thought might have the same effect in exciting coagulation as the addition of minute crystals or other fine particles to recently shed blood.

Thrombosis is so commonly a localised affection that we are forced to consider the local conditions which influence it as of primary importance. They may, so far as we know, be grouped under two heads—(1) changes in the nutritive condition of the vessel walls, and (2) changes in the regular equable flow of the blood. The changes in the vessel wall include all traumatic, inflammatory, and degenerative changes—however produced—and, of course, include the consequences of the invasion of the tissues by streptococci and other micro-organisms. It is when these changes affect the integrity of the inner coat, and particularly its endothelial lining, that coagulation is most prone to occur. I referred to this point in my last lecture, and we saw in the disease we were then considering—arteritis obliterans—a striking example of the influence of disease of the tunica intima in causing clotting. Atheroma, calcification of the middle coat of an artery when so advanced as to interfere with the nutrition of the internal coat, acute infections of vessels either from without or from the blood within them, and all forms of injury are frequent causes of thrombosis, and in many instances appear to be the sole causes acting independently of any special blood condition. However, we must not too readily conclude that a clot within a diseased vessel is caused entirely by that disease. In some cases an alteration in the blood is probably a contributory cause. The disease in the vessel is often very chronic, and coagulation may be the result of ill-understood cumulative conditions. The full vitality of the blood is only maintained when the circulation

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is vigorous, and all the great functions are regularly discharged. Diseases of blood-vessels are so very often associated with imperfections in the circulation, and with defects in one or other of the great viscera, that it must be that some change is often present in the blood flowing through these diseased vessels—a change predisposing it, perhaps, to develop platelets or to coagulate.

The circulatory disturbances which are believed to play an important part are those which result from (1) the sudden dilatation of a vessel, as one often sees in varix or aneurysm, or (2) the entrance of a small vein into a large one, or (3) the little pouchings naturally present in veins behind their valves. It is thought that in these situations slight irregularities of the flow of blood, little eddies, perhaps, may occur—what Recklinghausen has called "Wirbelbewegungen"—and that here blood corpuscles break up into platelets, and fibrin ferment is set free and clotting is started. In such a vein as the one I now hand round, where a dilatation of the size of a pullet's egg has developed in an internal saphenous vein which is not notably enlarged, we can understand that there must be a considerable departure from the regular even flow of blood in one direction—that is, from the normal condition. Within this great sacculus clotting has occurred, and it did not extend any distance beyond it. I excised this vein from the upper part of the leg of a woman, and with an entirely satisfactory result. Cohnheim held that the full vitality of the endothelium of a vessel partly depended upon the regular and rapid flow of blood over it, and he suggested that in such cases as those which I have mentioned, and in others of great enfeeblement of the circulation, the natural stimulus—or whatever influence it may be—of this rapid steady blood-flow is lost, that as a result the endothelium suffers, and that from this follows some deteriorating effect upon the blood leading to its coagulation. This view is interesting as emphasising the great mutual influence and dependence of blood and blood-vessel.

There are two modifications of the circulation that I would like to draw special attention to. The first is the regurgitation from deep into superficial veins, that is such a frequent, if, indeed, it is not an entirely constant, phenomenon in varicose veins. In the first of these lectures I described this phenomenon, and mentioned how constant I had found it. I believe that the disturbance in the circulation it causes, the undue tension in the varices, is an important cause of thrombosis in varicose veins. I make this statement the

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more confidently because, when I have operated in such a manner as to prevent this regurgitation (Trendelenburg's operation), I have found the tendency to thrombosis removed. I mentioned striking cases of this in my first lecture. I must not recapitulate them.

There is no doubt that thrombosis is much more common in varicose veins than in others. Of course, several other explanations of this fact are forthcoming, such as the effect of the abrupt dilatation and pouching that I have already referred to; the projection of the veins, exposing them to contusions and other injuries; and the degenerations or inflammations of the inner coat, which occur more often in varicose than in healthy veins. But the good effects of occlusion of the highest part of the superficial vein affected seem to me to be strong evidence that the greater tension and the modification in the blood-current caused by reflux must play an important part in leading to thrombosis. There is another well-known fact that bears upon this. I am sure that all of us have seen cases of thrombosis that we have treated *secundum artem* by rest in the horizontal position; we have been pleased to watch the arrest of the disease, the gradual lessening of the pain and swelling, and the passing away of all the unpleasant symptoms. At length the time has come when we have felt that the thrombus must be so far organised as to be firmly incorporated with the vessel wall, and that the danger of embolism is past. We allow our patient to get up; the legs that have been horizontal for some weeks, with low tension in all the vessels, are suddenly put in the vertical position, and the tension in all the vessels is raised. At once a fresh attack of thrombosis occurs. Why? From the injurious effect of an abrupt alteration (increase) of the tension in the veins. I have seen cases where this unfortunate accident has occurred time after time, until many months have been spent by the patient in resting for one after another of his attacks of thrombosis. But this clinical observation is such a commonplace one that I need not detail cases to illustrate it. We all of us, I suppose, regard the period of first getting up as one full of risk for a patient with thrombosis, so frequently is it followed by a fresh clot. Negative evidence to the same effect is afforded by the good effect upon thrombosis of rest in the horizontal position. How commonly it is attended with immediate arrest of extension of the clot and of all the other symptoms! It is the lessened tension in the veins that is the chief cause, I submit, of the improvement. And here I may mention one

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other point. We all know that thrombosis is much more common in the lower limbs than in the upper. Why is this? Is it not mainly because the tension in the veins undergoes greater, more frequent, and more prolonged increase in the lower limbs than in the upper? The general higher vitality of the upper than the lower limbs, which is shown in so many ways, may also partly explain this difference. A striking confirmation of this view is afforded by the fact that venous thrombosis occurs more often from tricuspid regurgitation than from any other form of heart disease.

The other alteration in the circulation that I wish to refer to is that produced by a block in a main artery. It is commonly stated that when a clot forms in a vessel it exhibits a tendency to extend in the direction of the circulation. This is undoubtedly true of venous thrombi, but it is not true to the same extent of arterial thrombi. Here we often see that the thrombus extends up along the artery towards the heart, and it may be for some distance. I may remind you of some of the cases of obliterative arteritis I mentioned in the last lecture, in which, for instance, the arterial disease with occlusion by clot extended as far as up to the popliteal artery, but the thrombus extended not only the whole length of the femoral artery, but in one case the clot reached up to the aorta, and even as high as the origin of the renal artery. I think it is truer to say that both in arteries and veins clots tend to grow centrally and towards the heart.

Another very important modification of the circulation is that brought about by great enfeeblement of the heart. Thrombosis undoubtedly occurs in many conditions of enfeebled circulation, and Virchow especially has described these so-called marantic thrombi. But not only is there no exact relationship between the rate of blood-flow and coagulation, but recent observations have demonstrated the presence of micro-organisms in many so-called marantic thrombi, and this discovery necessitates a careful reconsideration of our theories of intravascular coagulation from mere slowing of the blood-stream. Micro-organisms have been demonstrated in thrombi in many conditions, such as puerperal thrombosis, post-enteric, post-pneumonic thrombosis, and in the clots occurring in the late stages of tuberculous disease, as well, of course, as in the more definitely septic conditions. The organisms most commonly present in such clots are streptococci, staphylococci (less often), and occasionally the bacillus coli, bacillus proteus vulgaris, and some

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others. Only last year Dr. J. H. Bryant read a paper at a meeting of the Pathological Society of London in which he described four cases of thrombosis, where he found organisms in the clots which he regarded as the cause of the coagulation; in one case he found streptococcus and staphylococcus albus, in two cases bacillus proteus vulgaris, and in one case the bacillus subtilis in one saphena vein and staphylococcus in the opposite saphena vein, and in each case in pure culture. A great deal of attention has recently been paid to this investigation, especially by French observers, and the belief is gaining ground that micro-organisms play a great and hitherto unexpected *rôle* in the pathology of thrombi. It may be so. But possibly we shall take a too narrow view if, because organisms are found in clots or in the walls of plugged vessels, we at once assume that the organisms are the sole or main causes of thrombosis; indeed, I would venture to suggest that the whole discussion of the causes of thrombosis, of which I have given a very imperfect outline, is vitiated by one fallacy. The discussion is based on the assumption that a clear and sharp distinction is to be drawn between the blood and the blood-vessels. Do we not press this distinction too far? Developmentally we know that they own one common source; in the mature state their inter-relations are far more close than we readily imagine, and before we can truly appreciate the nature, causes, and conditions of thrombosis I think we must grasp the idea that the blood and the inner coat of blood-vessels are parts of one organ—of one organic whole. Any grave defect of this organ may lead to thrombosis, and whether the change is initiated in the fluid blood or the more stable tunica intima is not a distinction of primary importance.

Leaving these more theoretical considerations, I will ask your patience while I refer to one or two of the less common forms of thrombosis. I have seen at least two cases of thrombosis of the spermatic veins. One of the patients was a man, aged 32 years, under the care of my colleague, Mr. G. Lawson, at the Middlesex Hospital. On February 1st, 1894, he was kicked on the scrotum; a good deal of pain and swelling followed, but he continued at his work as an asylum attendant that day. Next day he stayed in bed and his pain was better. He tried to get up on February 3rd, but finding his pain worse he went back to bed and stayed there till February 7th, and after that he did a part of his work each day till February 14th, when he was admitted to hospital, because any

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strain or effort brought on an acute dragging pain in the left testicle. He was then found to have a firm, uneven swelling in the left cord in the upper part of the scrotum. The vas deferens could be traced over its posterior aspect, not lost in the swelling, and it seemed to be clear that this was a thrombosed spermatic vein. The patient was kept in hospital till March 5th, when he left convalescent. A year or two before this a youth, aged 18 years, was admitted to Middlesex Hospital under the care of Mr. J. W. Hulke with a swelling in the left spermatic cord. He gave no history of injury, and said that he had noticed a dragging pain in the part for three or four days. In this case a firm, narrow cylindrical cord could be felt extending from the top of the left testicle up to the external abdominal ring. It was clearly in the spermatic cord, and quite distinct from the vas deferens, and Mr. Hulke and others who saw it were quite clear in their opinion that it was a thrombus in a vein of the spermatic plexus. The testicle was a little tender only. There was no urethritis in either of these cases. This patient, like the other, was rested in bed, and left the hospital convalescent.

My colleague, Mr. Henry Morris, kindly allows me to mention a curious case that was under his care in the Middlesex Hospital. An unmarried woman, aged 28 years, accidentally noticed a lump of about the size of a pea in the left breast when she was feeling some pimples in the skin of the part. It gave her no trouble and she took no further notice of it. A year later, in May, 1899, she bruised the left breast, and two days after, on feeling the painful part, noticed a lump there which she thought was the same as the one noticed before. She was seen by one of the assistant surgeons to the hospital and advised to come in. The note of her condition on admission is as follows:—"A tumour of small size is situated in the outer and upper segment of the left breast towards the axilla. It is about one inch long and half an inch across, placed radially from the nipple. It is hard, smooth, somewhat lobulated; it is freely movable. It does not fluctuate. It is felt to be in the mammary gland itself, and to be over the pectoralis major. The axillary glands are neither enlarged nor hard. A large abnormal vein runs across the left axilla—a congenital abnormality." Mr. Morris operated and found the lump was a thrombosed vein in the breast. I think this must be a very rare experience, and with such a history as this patient gave it would

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have been almost impossible to arrive at a correct diagnosis before the operation.

I have seen some cases of chronic spreading thrombosis which, I think, are probably closely allied to cases of arteritis obliterans, and if I wanted to give a name to them I should call them *endo-phlebitis obliterans*. Up to the present I have only seen them in the superficial veins of the lower extremity. The patient notices a little stiffness in the leg hardly amounting to pain, and on feeling the part a slightly tender "knot" is felt. The symptoms at first are not severe enough to lead the patient to rest or to consult a medical man. Slowly the "knot" lengthens into a firm cylindrical cord, which can be rolled under the skin or may be fixed to it by periphlebitic œdema—it is only a little tender to the touch. Steadily this induration of the vein—for such it is—extends up the leg, and I have known it reach from the ankle to the groin. The connection of the firm cord with a subcutaneous vein can easily be demonstrated. So far as I have seen, the extension of the clotting and obliteration of the vein is always in an upward direction. I saw a case last year where a very robust, healthy, young married man, who had rather numerous but not very large varices scattered over the right leg and thigh, was attacked with this form of thrombosis. When I saw him he had already been many weeks in bed, and the clotting had been watched spreading up along the whole length of the internal saphena vein—quite up to the groin. The treatment of these cases by rest is very unsatisfactory—it may entail several months of idleness. Excision of the thrombosed vein, except at its earliest stage, is a rather troublesome and wholly unnecessary procedure. I have found that to excise a portion of the vein a little above the evidently diseased part effectually stops the spread of the trouble. This is a very simple procedure, and it has the further advantage that it removes the danger of embolism which might conceivably occur, even in these cases of chronic thrombosis. Three years ago I had a woman under my care with this affection, involving the left internal saphena vein up to a little above the knee. I removed an inch of the vein four inches higher up, and in 14 days she left the hospital convalescent, the disease being arrested and the pain to which it had given rise had quite passed off.

Among the cases of obliterative arteritis that I related in my last lecture was one that I was indebted to the courtesy of Dr. W.

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Pasteur for the opportunity of seeing and for the permission to publish. In addition to the disease in the arteries of the legs which led to gangrene in the feet there were acute abdominal symptoms during the last few days of the man's life, and it was to this abdominal condition that death was at last due. The symptoms were persistent vomiting, and the presence of an ill-defined tumour in the lower half of the abdomen—this was not resonant. The man died from exhaustion eight days after the onset of these symptoms. The necropsy revealed thrombosis of the superior mesenteric vein and partial obliteration of the companion artery; the mesentery and a certain length of the ileum were greatly thickened, hardened, and very dark in colour, being infiltrated with blood.

Thrombosis of the superior mesenteric vein is not a common condition; Dr. Welch has been able to find records of 32 cases. He states that the condition is commoner in the superior mesenteric vein than in the inferior, and much more frequent in the mesenteric arteries than in the veins. I had an example of superior mesenteric vein thrombosis under my care two years ago, and as the symptoms were exceedingly ambiguous I hope a brief summary of the case may be of interest. The patient was a carpenter, 40 years of age, who had enjoyed good health up to the time of the illness in question. This illness began 12 days before he was admitted to the hospital with languor and a sense of fulness at the epigastrium. His appetite was poor, and he ate very little. Three days later (May 17th) his bowels ceased to act, no flatus even being passed. Four days later still (the 21st) he came to the hospital out-patient department complaining of loss of appetite and constipation: he had no pain, his temperature was normal, and his pulse was 88. He was ordered sulphate of magnesia and haustus gentianæ alkalinus. After three days more (the 24th) he was seized with severe pain at the epigastrium, and for this he took next morning an ounce of castor oil which he retained, but which did not produce any action of the bowels. Then on the 25th he began to vomit, and he suffered much from paroxysms of griping pain at the epigastrium. On the 26th, as nothing had passed from the bowels for more than a week, his medical attendant administered a glycerine, and then a soap-and-water, enema which returned unaccompanied by any motion; he was now sent into the hospital. He was then, 12 days after the onset of the illness, a well-nourished man lying

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flat in bed with extended legs; he seemed to have no pain; his temperature was 98° F., and his pulse was 88; his tongue was thickly coated. His abdomen was not distended; it moved freely with respiration, its walls were flaccid; no peristalsis was visible. We could not detect any swelling or elicit any tenderness; the cæcum was not full. The liver edge was felt 2 inches below the costal margin at the end of inspiration; liver dulness was present. Elsewhere the abdomen was resonant. There was no hernia; the rectum was empty, and digital examination revealed no abnormality. The urine was acid, of specific gravity 1026, and loaded with urates; it was free from albumen, and did not give the indican reaction. Soon after his admission an enema of castor oil was given, followed by soap and water; this was returned without any motion, and soon afterwards he complained loudly of colicky pains, and his pulse rose to 108 and became very feeble. Hot fomentations soothed him, and in an hour his pulse was down to 80 and of good quality. The next morning (the 27th) Dr. Pasteur saw the man with me, and we decided to give him calomel in one-grain doses every four hours till the bowels acted. After two doses he passed a slight motion of dark, offensive liquid; he had great pain at the time and soon afterwards he vomited. But he had a comfortable night without pain, and next day he took an ounce of compound senna mixture. On the 29th the bowels acted twice, the motions consisting of a small quantity of dark fluid with a little solid fæcal matter. There was now no vomiting, he was free from pain, and slept well; his pulse was 96. His abdomen was still quite soft and moved freely; there were a little tenderness and sense of resistance to the left of the umbilicus; he was passing flatus freely. But he did not have a good night, and next day (May 30th) he complained of dull, aching pains in both loins; there was no vomiting, but also no motion; the abdomen was a little more distended, especially on the right side. At 3 P.M. he took half an ounce of castor oil, and afterwards passed two motions, chiefly fluid. He vomited in the night, and the acute pain returned. Dr. Kingston Fowler saw him with me on the next afternoon (May 31st) and agreed that there was no clear indication for immediate operation. We ordered morphia when the pain was severe. After this he became worse rather rapidly; the pain became very severe, vomiting returned, and his abdomen became distended and rigid, and his pulse rose to 144. In my absence

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Mr. T. H. Kellock operated next morning, June 1st. He found a quantity of blood-stained serous fluid in the peritoneum, a long coil of the small intestine was of a dark purple, almost black, colour, and the corresponding mesentery was thickened and firm with several hæmorrhages into it. Recognising that the condition was one not to be satisfactorily dealt with by operation Mr. Kellock at once closed the wound. The man died in the evening of the same day.

The following notes were taken at the necropsy: "Small intestine more or less gangrenous for entire length. Duodenum and upper one-third of jejunum were considerably discoloured, and below this the walls were almost black. The intestine was neither distended nor collapsed. Coils slightly adherent through early inflammatory exudation. Large intestine and vermiform appendix normal. Mesentery greatly thickened, firm, and yellowish-green in colour. Veins distended with blood-clot. Tissue round veins in portal fissure much thickened, and the portal vein contained clot. The chief vein of pancreas was distended and filled with clot, which, however, appeared more recent than that in mesenteric veins. No definite changes in arteries. No constriction or any other lesion to account for gangrene. Pancreas appeared rather swollen, otherwise normal. Spleen enlarged and distinctly firmer than normal."

Mesenteric thrombosis seems to be most often caused by some intestinal lesion, but in my case none was observed. The symptoms usually set in abruptly with severe pain of a spasmodic colicky kind, referred to the epigastrium; vomiting quickly follows, and the abdomen becomes distended and tender. There may be absolute constipation or there may be the passage of motions containing dark altered blood. Death appears to be an invariable result, and surgery offers no succour to these patients. I must confess to being entirely nonplussed by the case which I have just related; in several particulars it differed from the common course of the affection, especially in its slow, gradual onset, the length of time which it lasted, and the periods of apparent improvement in all the symptoms.

The last cases of thrombosis to which I will refer are two occurring in the innominate veins. The first was in a little girl, 10 years old, a patient under the care of Dr. S. Coupland in the Middlesex Hospital in 1894. She was admitted on December 18th

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suffering from typhoid fever. On February 11th, 1895, when convalescence seemed to be progressing well, each external jugular vein was found to be thrombosed—firm, knotty, and exquisitely tender; on the left side the swelling around the vein became very marked. A week later, February 19th, the note is: "Greatly improved. Feels well, and is recovering strength rapidly. Neck is excessively painful still, but inflammation is certainly subsiding. Both external jugular veins are thrombosed. Temperature 100°." On February 23rd she complained of pain in the chest below the right nipple. The superficial veins over the chest, especially on the right side, were enlarged, there was pitting on pressure on both sides of the chest, and the face looked puffy. There was no œdema of the upper limbs. There were no other physical signs of chest lesion. These symptoms continued, and on February 28th she had dysphagia. The œdema of the chest wall was greater; that of the face had disappeared. The neck was now less tender. The superficial veins over the chest were larger, and appeared to be carrying on a collateral circulation. Then signs of fluid in the left pleura were detected, and on March 8th she was tapped, and 24 ounces of semi-purulent and sanguineous fluid were withdrawn; this seemed to give a certain amount of relief. But the child got worse. Thus on March 12th the note runs:—"Child is generally worse; there is great puffiness of left side of face; great œdema of the chest wall. Pulse 136, very weak—indeed, scarcely to be felt at the wrist." The left pleura was again tapped on March 13th, and this time 1½ pints of yellowish cloudy fluid were drawn off. Under the microscope a few pus cells were seen in it, together with a large number of red corpuscles in various stages of disintegration. During the tapping the aspirator unfortunately broke down twice, and air was admitted to the pleura and to the subcutaneous tissue of the chest wall. Strange to say, two days later (March 15th) this child was sitting up in bed with a normal temperature and busy with her sewing. But the fluid reaccumulated, and on March 17th I opened and drained the left pleura, evacuating 2 pints of thin pus. For a time respiration was terribly embarrassed, and oxygen and brandy were administered; in an hour she became quiet, breathing well, and was evidently relieved by the operation. The patient then for a time steadily improved; her pulse became better and stronger, and we began to entertain hopes of recovery. On April 12th a trace of albumen

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appeared in the urine; this rapidly increased in amount, and by May 4th is noted as being present in "great quantity." At the end of April dyspnœa and restless nights returned, and on May 7th she was unable to lie down. There was then dulness at both bases up to the angle of the scapula. On the 9th I drew off 23 ounces of very sanguineous fluid from the left pleura; it gave immediate but partial relief, and she sank and died next evening (May 10th).

The great veins at the root of the neck are preserved in the Middlesex Hospital Museum, and the specimen is thus described in our catalogue:—"The veins of the neck and the superior vena cava. The superior vena cava is occupied by clot which projects into the right auricle. The innominate, subclavian, and jugular veins are completely blocked by clot and converted into fibrous cords. On the left side there is considerable matting about the upper end of the jugular vein. The azygos vein is enlarged. From a girl, aged 12 years, in whom the condition ensued five months after an attack of enteric fever. Double hydrothorax was present." (Specimen shown.)*

The second case was one that I saw four times in consultation with Dr. C. Percival White and Dr. Norris. The patient was a married lady, aged 30 years. During the seventh month of her first pregnancy accidental hæmorrhage occurred, and the child was born dead on May 12th, 1900. She passed through the puerperal state quite normally. At the end of four weeks she went away to Folkestone, and about a week later she developed a thrombus in the right internal saphena vein. Two months later—July 9th—she returned to London. Dr. White found her looking extremely ill, with a much higher pulse-rate than he could account for. Her temperature was 100° F.; the right saphena vein was still tender and firm. On July 16th she complained of pain in the right side of the neck. As there was an old-standing small thyroid adenoma here it was thought that this might be the cause of the trouble. But when I saw this patient again on July 19th there was no doubt whatever that clotting had occurred in the right axillary, subclavian, and external jugular veins—these could be felt as firm and very tender cords. There was marked venous congestion and œdema of the right hand and arm, and the patient complained of extreme pain in these parts. The œdema of the upper limb and right side of the

* 'Middlesex Hospital Medical Register,' 1155, 1893, P.M. 105, 1894.

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chest became enormous, but by the end of the month the œdema and the pain had both become less, although it was clear that the internal jugular vein was also plugged, and the patient complained much of buzzing in the ears as well as of the intense pain in the arm. The right arm gradually got perfectly well. Then the same process was gone through on the left side, but the pain and the circulatory disturbance did not seem to be quite so severe. At the end of August fluid was discovered in both the pleural cavities, and these were repeatedly tapped, clear serum being drawn off. The relief afforded was only transient, and on September 12th the patient died. The cause of the pleural effusions would appear in this case to have been venous engorgement only, due to obstruction of the azygos veins. In the former case there was evidently infection of the pleura with a pyogenic organism; it was not a hydrothorax, but an empyema.

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February 24th, 1902.

ON CARDIAC PAIN.

By E. Markham Skerritt, M.D. Lond., F.R.C.P.

It is so commonly our lot to have to deal with pain as an enemy, to be met with all the resources at our command, that this frequent contemplation of its gloomy side is apt to render us forgetful of its other aspect. In its inception pain is essentially conservative and protective. It is the sentinel warning of the approach of danger—the cry of Nature when attacked by foes who, thus discovered, may be repelled. In acute appendicitis pain has saved many lives; it calls for rest to an inflamed joint; it marks the occurrence of wounds, and watches over their repair; it warns the gouty man to mend his ways. Were it not for its protecting influence, many ills would befall us which we now escape. Some years ago I had under observation a hemianæsthetic man who met with accidents which his infirmity made of special interest. Once the garden wall fell upon him, and he found that he could not stand upon the affected leg, though it was free from pain. The bone was broken, and in the absence of pain might readily have been driven through the skin. Another day, when he went indoors after chopping wood, his wife found that he had lost the top of one of his fingers; he did not know that he had cut it off and left it in the garden.

Thus in its inherent nature pain is protective, and therefore beneficial. That it may appear in another *rôle* is an accident, and not of its essence, and happens when the cry for the removal of the cause cannot be responded to either by nature or by art.

Now cardiac pain, the subject of my paper, affords examples of pain in all its varied aspects and phases. And what has been said of pain in general holds good of heart-pain: it is in its essence protective and conservative, and tends to check strain that is fraught with danger. I do not propose to enter into a discussion of all its possible varieties, but rather to consider it from the clinical point of view as occurring under conditions determining stress upon the heart, of which undue strain it serves as a warning. I therefore exclude the pain of acute disease, and all pain of the nature of simple neuralgia or neuritis, which may occur

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in connection with the heart, as it does in association with other organs.

Pain that is the expression of the distress of the heart under too heavy a load of work is met with in all degrees of intensity, from slight and trivial discomfort at the one extreme up to the overwhelming agony of the classical angina pectoris at the other. There is no line of demarcation, either clinical or pathological; the distinction is one of degree only, and not of character.

The following clinical classification of the conditions under which such pain occurs may be adopted:—

(1) The heart is normal, but work is in excess.
(2) The heart is diseased—
  (*a*) Primarily in its valves;
  (*b*) Primarily in the substance of its wall.

In each case there is a disturbance of the balance between power and work, and pain is the cry of the overloaded heart.

Take first the group where the heart is normal, but work is in excess. This condition is met with in (*a*) simple overexertion; (*b*) the so-called vaso-motor angina.

(*a*) Pain may be caused in the normal heart by violent continued exertion, and is especially likely to occur if the individual is "out of condition," as far as concerns the particular form of exercise. This pain is referred directly to the heart, usually does not radiate, is slight or moderate in degree, commonly does not inhibit movement, and quickly disappears when the overstrain ceases. It has been met with, for instance, at the commencement of mountain climbing, and I have myself experienced it from hard riding when not in condition. If it is objected that it may be due to cardiac weakness or actual disease, I reply that it may occur where there is no other evidence of heart trouble, before the age of degeneration, and in an organ that remains free from suspicion. It is the outcry of the unfairly-burdened healthy heart.

(*b*) The other condition in which pain is associated with the healthy heart is vaso-motor angina. This is defined by Sir Douglas Powell, to whose writings on this subject I am much indebted, as a pure neurosis of the cardio-vascular system, a disturbance of the innervation of the systemic or pulmonary vessels, including sometimes the vessels of the heart itself, causing their spasmodic contraction, and thus increasing the peripheral resistance to the

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circulation. The sudden excessive demand upon the propelling power of the heart thus occasioned produces a more or less painful embarrassment of its action, which in some cases is increased by anæmia of the cardiac muscle, due to coincident contraction of the coronary vessels. It occurs mostly in early adult life, and about the climacteric period; in women of neurotic type, and in men from alcoholic and other excesses, and from tobacco poisoning. It may be induced by cold, by exertion attended with excitement, and especially by emotional disturbance of any kind. The nerves concerned are the sympathetic, the vagus, the depressor nerve, the vaso-motor nerves of the blood-vessels, and any nerve conducting afferent impulses to the nervous centres. The vaso-motor nerves have their centre in the medulla, and consist of vaso-constrictor and vaso-dilator fibres. The former pass to the vessels through the sympathetic, which is reached through certain anterior spinal nerve-roots and their *rami communicantes*; while the vaso-dilator fibres probably pass direct from the spinal cord through the anterior spinal nerve-roots without connection with the sympathetic. The currents through both sets of fibres are efferent. The sympathetic is the motor nerve of the heart, its stimulation being attended with waste and oxidation, causing acidulation of muscle. The pneumogastric is the restraining nerve, its stimulation being attended by regulation and restraint of action, favouring nutrition and repair. The depressor nerve is afferent, conducting impulses from the heart by way of the vagus to the vaso-motor centre in the medulla, whereupon the action of the vaso-dilator nerves causes a gradual but marked fall in the blood-pressure, largely brought about by dilatation of the abdominal vessels. The vaso-motor centres may be stimulated from the periphery, as by cold to the surface, or centrally through the emotions; and the sudden increase of intra-cardial pressure thus caused may embarrass the heart and find expression in cardiac distress and pain. As the intra-cardial pressure rises, the depressor nerve becomes stimulated in turn, and inhibits sympathetic activity; the vessels are relaxed, the pressure is relieved, and pain and distress disappear. Certain blood-conditions, especially the presence of uric acid and its allies, the absorption of ptomaines from the bowel, and the action of some drugs of the digitalis class, increase the general blood-pressure, and therefore make it easier for additional contraction of the peripheral vessels to cause acute cardiac embarrassment. Obviously fatigue

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and exhaustion of the heart as a muscle must attend the resulting laboured cardiac action; and Gaskell has shown that fatigue of muscle produces a tendency to cramp; and that accumulation of the products of waste in the heart muscle brings about an undue acidity which causes paralysis.

The symptoms of vaso-motor angina usually come on suddenly, with acute pain and a sense of oppression at the heart, accompanied by palpitation and dyspnœa; the pulse is generally small and quick, and shows the increased tension to which the attack is due; the surface is pale and cold, and the countenance is pinched. After a few seconds the pulse becomes relaxed, and the pain passes into a restless condition not met with in the graver form of angina. Between the attacks the heart is normal, except that some accentuation of the aortic sound may commonly be observed.

Sir Douglas Powell says that in its marked degrees vaso-motor angina is not very common; and that the prognosis is good, the younger patients, the climacteric cases, and a certain number of the gouty ones also, ending in recovery. My own experience would narrow the field in which this condition arises. I have met with it in gouty cases, and also at the climacteric period; but in the group of younger patients I have failed to recognise the vaso-motor element, and have been led to attribute the attacks to a more purely neurotic source. And what I have seen of the climacteric cases has led me to take a less favourable view of the prognosis, as their tendency has been to the persistency and increase of the abnormal tension, and the gradual development of corresponding cardio-vascular changes.

I do not include in this group (where the heart is normal, but work is in excess) the condition which obtains in Bright's disease, especially in its chronic forms, because in my experience cardiac pain is not induced while the heart remains free from degeneration. It is true that intra-cardial pressure is markedly increased, but the change is brought about so gradually that the left ventricle has time to accommodate itself to the altered conditions, and, by undergoing hypertrophy, to take on the extra work without distress. That the compensation thus secured is practically complete, is proved by the fact that the purest examples of hypertrophy of the left ventricle may be met with in chronic Bright's disease; always provided that the stage of degeneration of the cardiac muscle has not been reached, when the condition falls into Class 2, in which the heart is primarily weakened.

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In my second class, the element disturbing the balance between work and power is disease of the heart, occurring:—

(*a*) Primarily in its valves; or
(*b*) Primarily in the substance of its wall.

Valvular disease as a whole is not an important cause of cardiac pain. Authorities agree that the lesions most likely to be associated with cardiac pain are those of the aortic orifice. But it is a matter of surprise to me that there should be doubt or difference of opinion as to which affection of the aortic valve is more often the cause of pain; for in my own experience the one valvular lesion in which anginal symptoms are apt to occur in a marked form is aortic regurgitation. I find, however, that Walsh, a singularly accurate observer, speaking of the associations of angina, says: "In the case of the valves, aortic stenosis; very, very rarely insufficiency; and never, as far as I can recall, mitral disease, either stenotic or reflex." Sir Douglas Powell, while stating that "anginal seizures in association with aortic regurgitant disease are rather common," goes on to say that "in cases of aortic and mitral stenosis . . . . the ventricle is subjected to habitual strain in maintaining the circulation, and any extra exertion may overstep the limits of reserve power and bring about anginal symptoms"; and quotes a remarkable case recorded by Anstie of a boy who, while running sharply, suddenly cried out with severe pain and died; and *post mortem* great narrowing of the aortic orifice was found.

Now, of all valvular lesions, aortic stenosis is most completely compensated, and produces the least secondary effect upon the heart. In no other valve affection does such pure hypertrophy occur; and this is proof that the balance between work and power is here maintained as it is in no other instance. If, therefore, it is true that cardiac pain is due to disturbance of the relation of heart-power to work, it follows that it will least of all occur in aortic stenosis; and my own experience is in accord with the conclusion derived from theoretical considerations. Contrast what is met with in aortic regurgitation. Here the key to the situation is the occurrence of over-distension during diastole, which is the prime cause of dilatation. While the ventricle is relaxed in diastole, it has to receive both the blood entering from the normal direction and also that returned into it from the aorta, and it becomes dilated to contain the increased mass of blood. The ventricle,

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which also undergoes marked hypertrophy, forcibly expels its contents during the systole; but the very strength with which it overcomes the difficulty recoils upon it in the greater force of the regurgitation which follows; and hence events move in a vicious circle, hypertrophy reacting upon dilatation, and dilatation upon hypertrophy. When to this is added degeneration in the cardiac wall, as is practically always the case sooner or later, it is difficult to conceive conditions more likely to give rise to cardiac distress—closely analogous as they are to those existing when angina pectoris results from the association of increased arterial tension with weakness of heart-wall from degeneration.

If to the disability resulting from valvular lesion is added the factor of increased arterial tension—either temporary, as in the case of the vaso-motor angina before considered, or permanent, as the result of renal inflammation or primary vascular degeneration—it stands to reason that cardiac distress as expressed by pain will occur more readily than it would if disease of the valves did not exist.

My last division includes conditions where the heart-wall is the seat of degenerative changes. These are known to be of by far the greatest importance in the causation of angina pectoris. The morbid changes present may be first, fatty infiltration of the heart, independent of coronary degeneration; and secondly, those forms which are secondary to organic changes in the coronary arteries: which are, true fatty degeneration, fibroid hypertrophy, fibro-fatty change, fatty infiltration, infarction of the heart, and syphilitic arteritis extending to the cardiac substance. The essential pathology of the changes which are due to narrowing or occlusion of one or both coronary arteries is cardiac cramp and paralysis, and *post-mortem* evidence abundantly proves the overwhelming part played by disease of the nutrient vessels of the heart in fatal cases of angina pectoris.

When atheroma of the coronary arteries is marked, the balance between heart-power and work is proportionately disturbed, so that the enfeebled organ can no longer carry on the work of the normal circulation without giving signs of distress. A striking illustration of this came under my notice a short time since. A man, aged 62, had been unable for about two years to walk up even a slight incline, or to hurry at all on the level, without suffering from severe angina, the pain passing off when he stood

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still, to return at once on renewed exertion. The heart's action was rather weak; there was no definite cardiac enlargement, no definite accentuation of the aortic sound existed, and the radial pulse first felt exhibited a quite slight degree of thickening; but the other radial artery solved the problem, for it was the seat of a few rings of calcareous degeneration. The conclusion was that the coronary arteries were affected by the same change, and hence were unable to supply the needs of the heart-wall for any but the quietest work.

If, therefore, the weakening of the heart alone will so disturb the balance between power and work that the organ is unable to meet the ordinary demands of the circulation, how much greater will be the cardiac distress when the enfeebled heart is opposed by increased arterial tension? This may result from its association with the temporary vaso-motor spasm of the first group; but much more often the degenerated heart under consideration has to deal with a persistent increase in arterial tension, due to chronic nephritis, or to the vascular degeneration of advancing years. This is what usually occurs in the late history of chronic interstitial nephritis. The time comes when the hypertrophied ventricle, which hitherto has been able to hold its own with the increase of work due to the arterial tension, begins to fail in its nutrition, and of all conditions grave angina pectoris, with its risk of sudden death, is most imminent when the degenerate heart has to face increased obstruction in the arteries. The type of cardiac action indicates the danger which threatens. The pulse is usually quickened; it may be regular, or irregular and intermittent, and is often of good volume; but its notable feature is the high tension. The short, toneless first sound is hurriedly followed by a ringing aortic sound. This kind of action marks a ventricle that can hardly hold its own until the strain is taken off by the closure of the aortic valves; and it is plain that the patient lives on the brink of a precipice, over which he may at any time be impelled by a trifling increased demand upon the heart.

A few words on the interesting subject of the pathology of true angina pectoris. Various theories have been advanced, among which are the following:—

(1) That it is a neuralgia or a neuritis of the cardiac nerves. This view is held by Anstie, Eulenberg, and others. But the agonising cramp-like character of the pain, the suddenness of the onset, and the associated features, are unlike any neuralgic affection.

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And further, in those cases in which nerve lesions have been found *post mortem*, other conditions such as atheroma of the coronary arteries or of the aorta have also been present.

(2) Traube held the view that it was due to the great stretching of the nerves in the heart-wall, owing to extreme tension of the walls consequent upon acute dilatation, commonly associated with affection of the coronary arteries.

(3) Clifford Allbutt believes it to originate in the aorta, and to be due to aortitis.

(4) Osler has drawn attention to the theory of Allan Burns, as set forth in his work on the heart, published in 1809, that the condition is one of transient ischæmia of the heart muscle in consequence of disease or spasm of the coronary arteries. Burns quotes in illustration the condition known as "intermittent claudication." In the horse, and also in man, in consequence of thrombosis of the abdominal aorta or the iliac arteries, transient paraplegia and spasm may follow exertion. The collateral circulation, which is ample when the limbs are at rest, is insufficient when the muscles are actively used, and a state of relative ischæmia is induced, with loss of power, which soon disappears with rest. A heart with sclerotic or calcified coronary arteries is in an analogous state, and any extra exertion is likely to be followed by a relative ischæmia and spasm.

Sir Benjamin Brodie, writing in 1846, says:—"In examining the bodies of persons who died from angina pectoris, I have sometimes found ossification of the coronary arteries to such an extent that they were converted into complete bony tubes, while there was no disease of consequence elsewhere. When the coronary arteries are in this condition, they may be capable of admitting a moderate supply of blood to the muscular structure of the heart, and as long as the patient makes no unusual exertion the circulation goes on well enough. When, however, the heart is excited to increased action, whether it be during a fit of passion, or in running or walking upstairs, or lifting weights, then, the ossified arteries being incapable of expanding to let in the additional supply of blood which under these circumstances is required, its action stops and syncope ensues; and I say that this exactly corresponds to the sense of weakness and want of muscular power which exists in persons who have the arteries of the legs obstructed or ossified."

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Parkes Weber pointed out that the cramp pains and loss of power are both due to accumulation of waste products in the muscles; that they can both be induced in healthy persons by over-exertion; that they are more easily caused in persons whose vascular supply is inadequate, and whose tissue renewal is therefore sluggish; and, finally, that in angina pectoris from diseased coronary arteries the heart-failure may or may not be attended with painful cramp.

Gaskell has shown that the lymph fluid of all tissues at rest is alkaline, and that the natural activity of muscle is always accompanied by dilatation of its blood-vessels; and argues that this dilatation is brought about by the diminished alkalinity of the lymph fluid of the tissue during its activity, which relaxes their muscular coats. When the vessels are narrowed and rendered rigid by disease, the arrangement for flushing the tissues with blood while in action is interfered with, and hence the production of the phenomena of paralysis and cramp met with in angina pectoris.

Time fails me to speak of the treatment of cardiac pain, except to refer to its bearing upon the fact that heart-pain is caused when the balance between power and work is disturbed. This loss of balance can be remedied in one of two ways: (1) By increasing the power of the heart, or (2) by lessening the work which the organ has to do. And in practice we find that the latter is usually the easier course; and we restore the balance for the time being by diminishing vascular tension by means of the vaso-dilator remedies.

Dr. Symes Thompson congratulated the author on his practical paper, but expressed disappointment that the page on therapeutics was so short. But one might be less sorry for that, because Dr. Skerritt had clearly indicated the lines of treatment which were likely to be of service, and it was more satisfactory to have the mind directed to the precise conditions giving rise to pain, rather than to have a series of narcotics and sedatives suggested for temporary alleviation. He was not prepared to traverse the suggestions put forward, which were exceedingly valuable, and the author's sub-division of the subject was calculated to give much help. He would have been prepared to hear a little more about the functional, and a little less, in proportion, about the organic, diseases giving rise to pain, because physicians were accustomed to meet with many serious pains in the cardiac region which were found to be dissociated from, or independent of, any appreciable organic change. He supposed the heart would never have been regarded as the centre of the emotions but for the fact that it was so freely supplied with nerves from various sources, which were very apt to be placed out of gear by emotional influences. He would confine his few remarks to the pains met with in hysterical

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and hypochondriacal subjects. These pains were to a large extent dependent upon constitutional conditions at the particular moment; that which would at one time give rise to pain, would not do so under more favourable conditions of nutrition and happiness of mind. One met with so many cases of functional pains that it would be advantageous to devote some consideration to them. He was reminded by the opening sentence of the paper of a somewhat curious case, that of a young medical man who, impressed with the belief that he had very serious cardiac disease, consulted him frequently. Nothing of the kind could be detected, but the patient still believed that he (Dr. Symes Thompson) was wrong. One day after smoking he put his pipe into his breast pocket and then went cycling. Shortly afterwards he became conscious of a very acute pain in the cardiac region, and was congratulating himself on being able to prove to his doctor that he certainly was wrong, when a small boy drew his attention to the fact that his coat was on fire, and that the pain did not arise from organic disease. It was important to bear in mind that very many pains of a seemingly serious character were due to hypochondriacal and hysterical causes.

Dr. SANSOM said that Dr. Skerritt's paper would be of great value to the profession generally. He, with Dr. Symes Thompson, hoped to have learned a great deal with regard to clinical forms of cardiac pain. The author had confined rather narrowly the term cardiac pain in a way which perhaps was not clinical, but rather scientific at times. Some pains were distinctly referable to the cardiac mechanism, which, at others, could not be thus accounted for. The author instanced a form of angina which was strictly not pain, the "angina vasomotoria" of Nothnagel, which was regarded as sometimes the outcome of over-exertion. Physicians saw many people who had been great at athletic sports, but only few persistent cases of heart pain were met with among them. It was generally agreed that the first sign of trouble was in the right heart, but it was very astonishing how soon the right heart accommodated itself to the altered circumstances, and the patient got well. He thought the "angina vasomotoria" might be ascribed to the distension of the right heart. He thought over-exertion was credited with more mischief than was its due. On the other hand, he had seen much good result from systematic exercise in cardiac diseases. Some years ago he placed before the Society the results of his observations on the left side of the heart, and expressed his belief in such a condition as athletic hypertrophy. Athleticism produced in many a considerable increase of power in the left ventricle, which could not be ascribed to morbid conditions, and was not in itself a harmful condition.

With regard to the cases of heart-pain which came before physicians in hundreds, a great many of them were to be ascribed to causes extrinsic to the heart. There were the neuralgias and neurites—he did not believe in such a thing as rheumatic neuritis, though he did in gouty neuritis—and there were many cases in which digestion was affected, probably from zymotic causes. Extrinsic causes of cardiac pain might be much mixed up with other conditions, and he thought they might conclude that at least 50 per cent. were due to causes which were extrinsic to the heart. With regard to the cases in which the heart itself was involved, he confessed to a change of front. In times past he used to say that wherever there was true angina pectoris it was associated with thickened arteries, *i.e.*, some signs of arterial sclerosis. In fact, he had said that if the arteries were not thickened it was not true angina pectoris. He had

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since been shaken in that view. Huchart collected hundreds of cases in which coronary atheroma was associated with angina pectoris. He knew now there were cases of angina without any sign of thickened arteries, and Dr. Arnold, of Rugby, was a famous example. He had seen a lady of advancing age in whom he could find no sign of either hypertrophy of the left ventricle or of thickened arteries, but who had signs which were considered by him and others as those of angina pectoris. He thought she would recover, but she died. There was no thickening of arteries, but there was anuria, and that, he thought, was the sequence which caused the death, by contraction of the arterioles of the kidney. He had since leaned to the idea that ischæmia cordis from the contraction of arterioles had much to do with anginal attacks. These were probably due to complex nerve conditions, and he differed from Dr. Skerritt in that he thought much was to be said for the immediate nerves involved. In one of the cases observed by Lancereaux there was distinct neuritis, and the affection of the aortic valves bared some of the filaments of the nerve. He agreed that the valve lesion most associated with angina was aortic regurgitation and not aortic stenosis, the latter being very rare. He believed that in aortic regurgitation there was a fraction of a second in which the strain was very great, and intense pain might thus be initiated.

He believed in the paramount duty of giving relief from pain, whether it occurred in real angina or in the simulated forms. In the milder forms the slighter remedies were the best, *i.e.*, phenacetin, antipyrin, &c., rather than the opiate remedies, though he agreed that in some cases opium must be used, but then as sparingly as possible, and preferably by hypodermic injection. With regard to the employment of arterial relaxants, such as nitrite of amyl and nitro-glycerine, they were very valuable, but, like morphia, were liable to abuse. He had seen cases of patients with relaxed tension who had swallowed half-a-dozen nitro-glycerine tablets without there being any reason for it. He believed a very useful tabella had been originated at Westminster Hospital containing nitro-glycerine and nitrite of amyl to be swallowed, and he had seen much good result from its use.

Dr. De Havilland Hall regretted he had not heard the whole of the paper. With reference to the tabella, the origin of which Dr. Sansom rightly ascribed to Westminster Hospital, as it was introduced by his colleague Dr. Murrell, it had been discarded because the nitrite of amyl was found to be so volatile that it was useless. He believed Messrs. Burroughs and Wellcome carried out Dr. Murrell's idea, but they also had omitted the amyl nitrite recently, and were sending out menthol and nitro-glycerine. He wished to narrate a case which very much impressed him. A gentleman of between 50 and 60 years of age consulted him more than 20 years ago on account of pains, which he (Dr. Hall) considered to be of the nature of angina pectoris—excruciating pain in the region of the heart, accompanied by much depression and faintness. Neither nitro-glycerine, which had recently been introduced into medicine, nor nitrite of amyl gave relief. He was, however, much benefited by a course of blue pill and mistura alba. As the patient got restless and fidgety, Dr. Hall saw Dr. Andrews, who was then senior physician of St. Bartholomew's, about the case. He agreed the patient had angina pectoris, but had no suggestions to offer with regard to treatment. The pain continued for three or four months, and sometimes was very severe. At length he was called to see the patient in the country, in consultation with his medical

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attendant, and found him apparently suffering from hepatic colic. He was very jaundiced, and the attack was followed by a hepatico-bronchial fistula. After that the attacks of so-called angina ceased, and he had no other until he died seven years afterwards. Two or three years later he saw an article by the late Dr. Brinton, in which he said that no doubt many cases of obscure pain in the region of the heart which had been attributed to angina pectoris were really due to hepatic colic.

The PRESIDENT said he did not share the feelings of disappointment expressed by two speakers in regard to the paper, because he understood from the title that it would only deal with those forms of discomfort and pain which were of purely cardiac origin, and would leave for other consideration those, perhaps more numerous, which were purely extra-cardiac in character. In reference to pain of cardiac origin, there was some little difficulty in the detection of it, partly from the variation in the actual situation of the pain. It was a matter of great interest to him as to how far pains were actually located in the heart, even when there might be very obvious cardiac cause for them. He thought it was commonly the case that the actual pain was less in the heart itself than in some of the areas which were nervously connected with that organ. The actual pain might extend over a considerable region.

In a large proportion of cases the pain of structural heart disease was referred to some distance from the heart, and notably in those severe cases in which the pain was felt in the left arm. With regard to the nature of the pain itself, it was noteworthy that most changes in the viscera often gave rise to very little pain until the serous membrane covering them was extensively concerned. That was the case in both abdominal and thoracic disease. The most constant circumstance in connection with the pain of cardiac origin was the condition of actual degeneration of the viscus. The most severe pains were those in angina pectoris, in which there was always some amount of degeneration, associated with anæmia of the heart itself from disease of the coronary vessels, and this same condition it was that brought about sudden death by the production of fibrilar contraction or delirium cordis, which experimentally had been shown to depend upon an anæmic state of the heart. At the same time it must be admitted that many cases of cardiac degeneration were to be met with which were quite free from pain. The question whether the cause was aortic obstruction or aortic regurgitation he regarded as minor compared with that great main fact, and it was rather the question to what extent either of those particular lesions was associated with a degenerative state of the heart. In a large proportion of the cases the condition of heart disease, which led to the existence of a systolic murmur at the base connected with the aorta, was one in which the valves were considerably degenerated and in which there was evidence of disease of coronary vessels, and some degree of degeneration of the heart itself, and hence, although aortic stenosis was relatively infrequent, he believed that angina pectoris was more often associated with a basi-systolic bruit than with a diastolic one. But it was very difficult to explain exactly what the intimate nature of the pain was—whether neuralgic, neuritic, &c. He was surprised to hear Dr. Skerritt on two occasions refer to cramp in connection with the heart. One knew definitely that the heart-muscle presented a very great exception to other muscular tissues in the body, in that the condition of cramp or spasm of the heart was non-existent. The pain occurring in systemic muscles elsewhere, and in the plain

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muscular tissue of the stomach or intestines, was very acute, and of the nature of cramp, but spasm of the heart could not exist. Although Dr. Skerritt had so clearly set forth the actual bearings of the question, it was still left to the future to explain the actual intimate nature of the pain itself.

Dr. MORISON said the subject was so wide that the paper might be a synopsis for a series of lectures; yet when these had been finished, the subject would probably not be at an end. He approved of the limitation of the paper under these circumstances to certain points, and especially because it excluded treatment which required to be fuller to be satisfactorily discussed. He differed, with all respect, from the President in regard to the appropriateness of the term cramp as applied to the heart in these cases. There could not of course be a general cramp of the heart without its coming to a standstill, but he thought it was nevertheless a felicitous term to use in regard to true anginal attacks. The effect of such a cramp was to produce inhibition of the heart's action, which, in fatal cases, terminated in syncope. He thought evidence of such a condition could with some probability be found microscopically *post mortem*, in the condition known as muscular fragmentation, which was observed in fatal cases. That that was not associated with absolute rupture of muscle during life was evidenced by the fact that it did not show effusion of blood in the broken tissue. He thought, however, that *localised* cramp, leading to inhibition and syncope, was one of the main causes of pain and a fatal issue in many cases. He thought the originating cause of the pain of angina pectoris was largely in the heart itself. Some years ago he had seen an instructive case of pulmonary obstruction and regurgitation associated with anginoid pain, the pain being felt in the right arm, and the lesion being limited to the right side. This case was examined also after death. He agreed with Dr. Skerritt that it was mostly in cases of aortic regurgitation that angina pectoris was met with when associated with valvular disease, for rarely did one see well-marked aortic obstruction with calcareous valves driven dome-shaped into the aorta; but he had observed well-marked angina pectoris in such a case. He said that if one made transverse sections of a coronary artery and stained the nerves, one was impressed by the rich nervous endowment in the neighbourhood; and though it might be heresy to say that, in cases of calcareous coronary arteries, it was possible that a heart thus crowned with a rigid tube was in any way provocative of anginous attacks by direct irritation of such neighbouring nerves, he could not disabuse his mind of the idea that in some cases this was so.

Dr. PARKES WEBER said he understood that the President thought it was impossible to get cramp of the heart. But he (Dr. Weber) would have thought that the mere fact of the heart being a muscular organ was sufficient to prove the possibility of cramp in it; indeed, cramp could occur even in unstriped muscular organs. Surgeons had occasionally in laparotomy cases seen parts of the intestine in a state of cramp. He thought the supposition of complete cramp occurring in the heart would account for some of the fatal cases with a short period of cardiac pain preceding death.* In some of the more prolonged cases of angina

* Likewise for some cases of fatal syncope, apparently *without any preceding cardiac pain.*

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there might be a condition of localised cramp. One of the speakers had referred to an irregular muscular movement producing a "fluttering" action of the heart, but why should not sometimes another kind of irregular muscular action occur—namely a localised cramp in a small portion of the heart muscle, changing, perhaps, from one spot to another? Persons who had often experienced universal cramp in a group of skeletal muscles knew that certain painful sensations indicated the presence of localised cramps, often followed by complete cramp; they sometimes knew how to prevent the generalisation. With regard to the waste product theory of angina pectoris, and of the painful cramps of limbs in which the arterial supply was obstructed, the point was that, owing to the defective blood stream, there was a lack of flushing out of the waste products resulting from muscular exertion. With regard to the skeletal muscles, in even healthy people there was often a tendency to the occurrence of sudden cramp every now and then for some time following severe muscular exertion, and that fact, he thought, could well be explained on the accumulation of waste-product theory.

Dr. Pasteur said he had been struck by the infrequency of actual pain, as opposed to distress or discomfort, in disease of the heart. He thought that would be the experience of everyone with a large experience of such cases. Everyone would agree that aortic insufficiency furnished the largest number of cases presenting cardiac pain, but, excepting these, the most striking he could recall were those in which there was degeneration of the heart-muscle. In these cases the pain, which was not necessarily of anginal character, was most wearing and prolonged. It was well to remember that pain in the heart was, fortunately, an uncommon event.

Dr. Rolleston.—The question of "cramp" of the cardiac muscle as the cause of angina pectoris was extremely attractive, but it must be admitted that if the cramp was universal death must ensue, and this explanation would only explain cases where death occurred during the paroxysm. If cramp of the myocardium occurred in the repeated seizures of true angina pectoris it must be partial, and only involve a limited part of the heart-muscle. The suggestion was plausible that the cramp affected the musculi papillares only, while the remainder of the ventricular-wall went on contracting and carried on the circulation. The musculi papillares were furthest from the coronary arteries, and therefore received a less satisfactory blood supply; further, accumulation of toxic products, due to metabolism, would more readily take place in them under conditions of imperfect blood supply, such as obtained in most examples of true angina. He quite agreed with Dr. Pasteur that, apart from angina, true cardiac pain was rare in hospital patients; it undoubtedly occurred in acute dilatation of the ventricles, in embolism of the larger branches of the pulmonary artery, and in that rare condition—embolism of the coronary artery. The diagnosis between true angina and vasomotorial angina in young people was often very difficult. He remembered a case of a man, aged 23 years, who had attacks of angina accompanied by great flatulence and nervous symptoms, and who might have been regarded as an example of vasomotorial angina; sudden death, however, supervened, and a localised patch of atheroma at the base of the aorta, obstructing the orifices of the coronary arteries, was found. In true stenosis of the aortic valves with a mere chink-like orifice—a rare disease—atheroma at the base of the aorta, contrary to what might be expected, was seldom seen. This fact might account for the comparative infrequency of true angina

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pectoris in true aortic stenosis. It was worth noting that atheroma of the trunks of the coronary arteries, though it rendered them more or less rigid tubes and incapable of further dilatation, did not always or necessarily narrow their lumen. In some instances of endarteritis deformans the trunks of the coronary arteries were even larger than natural; this accounted for the absence of angina in many cases of atheroma of the coronary vessels. Fragmentation of the heart-muscle, which had been chiefly described in America, was a condition about which he was doubtful. As a result of microscopic examination of a number of hearts, he was not convinced that the appearances represented changes that had taken place during life. Further, it was unlikely that fragmentation could be regarded as proof of the "cramp" of angina pectoris, since the hearts practically always underwent rigor mortis before examination could be made, and it must be admitted that if cramp produced this fragmentation rigor mortis might do so too.

Dr. Campbell Pope said he would be glad if Dr. Skerritt, if he had time, would say expressly what he meant by cardiac pain. One speaker had mentioned pain on the right side radiating down the right arm, and commonly pain radiated down the left arm. There were cases in which there was fairly acute distress on the right side of the chest, and many in which the pain had been referred to the left axilla. Last Friday night he was called to see a man who had come into a legacy, and whom a nephew whom he had not seen for some years wished to visit, but the man wanted to prevent his visit, and excitedly, by words, disputed his entry. He fell down dead. Next day he, Dr. Pope, found a chink-like orifice of the aortic valves, considerable atheroma in the aorta, and the coronary arteries were like tobacco pipes, and with a very much obstructed lumen, through which a small probe could not be passed. There was no history of angina in that case, showing that angina did not always go with those cases. Perhaps the minor forms of cardiac pain came more before the general practitioner than before the hospital physician, and he had seen cases due to tea, especially when combined with the eating of cake and the taking of much sugar. In some such cases there was no pain when tea was taken with breakfast, but after further supplies of it during the day the persons could not walk 100 yards on account of the pain. He was interested in hearing of the tabella which had been mentioned, but he thought that there was a danger of forgetting the good old remedies, especially the Liq. Ammon. Acet. with Spts. Ætheris Nit., for relaxing vascular tension.

Dr. Markham Skerritt, in reply, said the hour was too late to answer adequately all the questions which had been put to him. With regard to the situation of the pain, that was variable. In many conditions there was pain in the heart, and in others there was reflected pain, which latter was very uncertain in distribution. For instance, cases were on record in which the pain was felt in most unlikely places, such as in the testicle and in the hip joint. The commonest situation of reflected pain was down the left arm, which probably would include the left axilla. The question of the association of other areas of pain with heart affections was a very interesting one, but it was impossible to go fully into the question. Certainly bad pain was rare in heart disease. With regard to Dr. Weber's remarks, he intended to express the difficulty there was in flushing the muscle in action, owing to the rigid condition of the coronary arteries. He agreed that one might meet with

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athletic hypertrophy, without its being a disease in the general acceptation of the word. It was the result of great and prolonged sytematic muscular exertion, just as the hypertrophy of any other much-used muscle might be. He was very much indebted for the reception of his paper, and for the valuable discussion it had created.

A NOTE ON HYPOSTATIC ALBUMINURIA OF SPLENIC ORIGIN.

By H. D. Rolleston, M.D. Cantab., F.R.C.P. Lond.

This short communication deals with a small point which has very probably often been observed before, but being an isolated observation has not been thought worthy of much notice. The point to which I wish to draw attention is that in some patients with considerable splenic enlargement, rest in bed or in the recumbent position may be accompanied by albuminuria, and that the albumin may disappear from the urine when the patient assumes the erect position. This fact was first brought to my notice in a case of advanced leukæmia, which I often saw with Dr. C. J. Heaton, of Westgate-on-Sea, who drew attention to the intermittent albuminuria, and explained it on Falkenheim's* suggestion that in the recumbent position the spleen pressed on the left renal vein, interfered with the return of venous blood from the kidney, and thus gave rise to albuminuria. This seems to be a very reasonable explanation of the occurrence. I have recently had under my care two other patients, with hepatic cirrhosis and splenic anæmia respectively, with considerable splenic enlargement, presenting a rather large quantity of albumin in the urine while lying in bed, which disappeared after they had been up for several hours. The albuminous urine was often high coloured, and, like the urine of backward pressure in cardiac disease, lithatic. In all my three cases the night urine was sometimes found to become free from albumin, possibly as the result of changes in the position of the body such as had been studied by Falkenheim.

It is, of course, necessary that the bladder should be emptied of urine when the patient gets up, otherwise an admixture of the

* Falkenheim ('Deutsches Archiv für Klinische Medicin,' Band xxxv, S. 446, 1884) described exactly this condition in a man, aged 50 years, with hepatic cirrhosis and a large spleen. He observed that lying on the right side and on the abdomen did not induce albuminuria.

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albuminous urine, excreted while in the recumbent position, with the normal urine of the erect position will occur, and the patient may be thought to have continuous albuminuria. The existence of this hypostatic albuminuria, if the term may be used, should be borne in mind when dealing with cases where, as in hospitals, the sample of urine provided for examination is taken from the whole amount passed in the 24 hours, or is that passed early in the morning while the patient is in bed.

While the albuminuria is probably due to the mechanical pressure of the spleen on the left renal vein in the recumbent posture, the occurrence of this intermittent or hypostatic albuminuria is very far from constant in cases of splenic enlargement. In this it resembles albuminuria from chronic venous engorgement induced in other ways, as, for example, in the backward pressure of mitral disease. It does not depend on the size of the spleen, for hypostatic albuminuria may be, and, indeed, usually is, absent when the spleen is very large, and may be present, as in two of my three cases, where the spleen is relatively smaller. It is possible that its occurrence or absence may depend on some difference, such as elongation, in the condition of the suspensory peritoneal ligaments of the spleen which thus determine or prevent direct pressure on the left renal vein. On this point there is as yet no anatomical evidence. It is also conceivable that the appearance of albuminuria is dependent on an underlying want of vitality or nutrition in the kidney which, though not sufficiently marked to induce albuminuria under ordinary conditions, does so when chronic venous engorgement is superadded. This is to some extent supported by the observation that in one of the more recent cases albuminuria disappeared from the night urine after the patient had been some time in the hospital, and had somewhat improved under treatment; subsequently, after a slight attack of influenza, the night urine again contained albumin.

It appears that the appearance of albuminuria in the recumbent position, and its disappearance in the erect position, is not absolutely constant even in those individuals with enlarged spleen in whom its presence has been noticed; but its temporary occurrence in three cases justifies the suggestion that it may not be so very infrequent.

This hypostatic albuminuria is the reverse of what usually occurs in cyclical albuminuria, but it is possible that there may be some cases of cyclical albuminuria in which albumin appears when the patient is lying down and disappears when he is up. I have not

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yet met with a case, but the possibility of such an event is suggested by Edel's* recent observations that a weak pulse and the appearance of albumin in the urine in cyclical albuminuria are related, and that if, by regular muscular exertion, such as marching or hill-climbing, always stopping short of fatigue, the heart's action is strengthened, the urine becomes free from albumin, lighter in colour, and more copious.

CASE 1.—Male, aged 43. Spleno-medullary leukæmia. First came under observation complaining of passing uric acid. Great enlargement of spleen. Albuminuria was noticed when he lay down, and was absent in urine passed after he had been up for some hours. On one occasion at least Dr. Heaton found the night urine for several days running free from albumin.

CASE 2.—Male, aged 43. Cirrhosis of liver. Rapid onset of ascites after hæmatemesis in July, 1899. He was treated by the operation of opening the abdomen and producing artificial peritoneal adhesions, and was shown by Mr. Turner and myself in December, 1899, at this Society.† In January, 1902, he came into St. George's Hospital with abdominal distension, some ascites, œdema of the legs, and splenic enlargement. When lying in bed his urine contained a heavy cloud of albumin, when he was up all day urine taken at the end of the day was free from albumin. Subsequently when confined to bed by a severe attack of phlebitis his urine remained free from albumin for a time, but albuminuria reappeared later on.

CASE 3.—A girl, aged 17. With splenic anæmia. Admitted into St. George's Hospital in January, 1902. On admission it was noticed that when she remained in bed the urine contained a heavy cloud of albumin, the urine passed after she had been up for some hours was found to be free from albumin. Subsequently the urine became entirely free from albumin for a time, later albuminuria on the recumbent position recurred.

Dr. SANSOM said it must not be forgotten that there might be something behind the enlarged spleen, that it might be the outcome of something else which helped to produce that postural albuminuria. Teissier had made a study of postural albuminuria, and it was probable that one form of postural albuminuria might be due to the spleen. Then behind the enlarged spleen there might be malarial or other affection, such as influenza. One might have gone too far in times past in attaching significance to albuminuria. He was referred to in the case of a young girl abroad, who was a source of revenue to analytical chemists in the vicinity, but about whom there was nothing organically wrong. Still, the observations of Dr. Rolleston were very valuable as exemplifying the condition which Teissier had worked at.

* 'Münchener Medicinische Wochenschrift,' 1901, Nos. 46, 47 (Abstract, 'Brit. Med. Jour.,' 1902, vol. i, epitome No. 63).

† Rolleston and Turner, 'Trans. Med. Soc.,' vol. xxiii, p. 63.

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The PRESIDENT said he would have thought, in a case such as the present, it was absolutely necessary to exclude such possible explanation of the albuminuria as the blood condition which might be responsible for it. The position of an enlarged spleen in reference to the kidney he thought was not quite such that one would have expected it to press upon the left renal vein, bearing in mind the direction in which splenic enlargements generally went. But it would need to be demonstrated *post mortem*, where the spleen was very large, how the kidney became mechanically congested.

Dr. ROLLESTON, in reply, said that of course there was some condition underlying the splenic enlargement in his cases—viz., leukæmia, hepatic cirrhosis, and the cause, whatever it was, responsible for splenic anæmia. But these underlying conditions did not serve to explain albuminuria in the recumbent position and its absence in the erect position. As a matter of fact malaria was not present in his cases; but even if it had been, it would not explain the intermittent type of albuminuria. There was, of course, a recognised malarial nephritis, but in nephritis there was a tendency to greatly increased albuminuria in the erect position as compared with the recumbent position. He was not acquainted with Teissier's cases of postural albuminuria.

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March 10*th*, 1902.

REMARKS ON A SERIES OF CASES OF EXTERNAL OPERATIONS ON THE LARYNX.

By A. Marmaduke Sheild, M.B., B.C. Camb., F.R.C.S.

The operation of thyrotomy is one of considerable antiquity, though we question whether its full scope and utility have been recognised until within the last few years. The late Mr. Durham stated that Ehrmann, in 1844, first performed this operation for laryngeal growths, which he diagnosed, of course, without the aid of the laryngoscope.* Since then a number of operations upon the air passages by external incisions have been performed, particularly in Germany, and Hahn especially greatly improved the technique of the operation of thyrotomy, introducing the well-known inflatable tracheal tube which bears his name.

The Operation.

The following series of cases seem of sufficient interest to warrant their publication, as they illustrate the scope and excellent results which are to be obtained by thyrotomy and other external operations upon the air passages. As a preliminary I would describe the method of operation, which differs in some small particulars from that usually adopted. In the first place, the services of a highly-skilled anæsthetist are essential, and no surgeon should attempt one of these cases without such aid. Chloroform is, I think, the best agent to employ. A good light is needful, and in dark weather the aid of a forehead electric lamp is often highly advantageous.

When I first commenced to perform these operations, I followed the usual rule, and made two incisions: one for the preliminary tracheotomy, the other for the division of the thyroid cartilage. The somewhat limited area thus exposed rendered the checking of all hæmorrhage very tedious, and thus materially increased the difficulty and length of the operative procedures. I now strongly advise that the larynx and trachea should be exposed by one longitudinal incision from above the thyroid cartilage to a point

* Holmes's 'System of Surgery,' 2rd edition, vol. xi, p. 697.

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1½ inches below the cricoid. This incision is deepened until the cartilages are reached, and on retraction of the edges of the wound the whole field of operation is laid bare in a remarkably advantageous manner. The hæmorrhage is always profuse, but in this large wound the veins are caught with pressure forceps, and tied or clamped with ease and alacrity. Indeed, the checking of hæmorrhage by this method effects an agreeable contrast to the small incision, where the perpetual "welling up" of venous blood is a serious inconvenience to the operator. At the lower extremity of the wound the thyroid isthmus is seen, and this may be drawn downwards, so as to uncover an additional ring of the trachea; if large it may be divided between two clamp forceps. During all these proceedings the patient's head is well thrown back and steadied by an assistant.

The second stage of the operation—that of opening the trachea—has now arrived. This is done easily and deliberately below the cricoid cartilage, and the tube is inserted. Personally I prefer the tube covered with compressed sponge. This I powder with iodoform before introduction. I usually allow eight minutes to elapse for the sponge to swell and the anæsthetist to adapt himself to the new conditions of administration. The methods of administering the chloroform vary with different authorities. Personally, I prefer the administrator to blow the vapour into the tube by means of a small bent cannula. This allows any blood or mucus to be instantly cleaned out of the tube by means of feathers or a brush. It may here, too, be pointed out that the tube, whether covered with compressed sponge or surrounded by the usual inflating rubber bag, should be tested by the surgeon with scrupulous care, to ascertain for himself that the accessory materials are properly secured. When the sponge, for example, swells in the trachea, some difficulty is experienced at the end of the operation in withdrawing the tube from the tracheal wound, and if badly constructed, the sponge may easily become detached—an awkward and alarming accident. All brushes should in like manner be well made, so that any possibility of their heads "coming off" is out of the question. The tube being secured by tapes, if the thyroid cartilage be calcified, as is common in adults, it may be divided by a fine saw—a very fine saw *—or by strong scissors curved on the edge. If possible, the extreme upper border of the cartilage should be

* The blade should be flexible and the teeth minute.

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left intact, so as not to interfere with the petiolus of the epiglottis. The assistant now grasps each cut margin with artery forceps and thoroughly everts and retracts them. Some surgeons prefer sharp hooks for this purpose. The growth or foreign body at once comes into view in a remarkably clear manner. If a growth, the novice

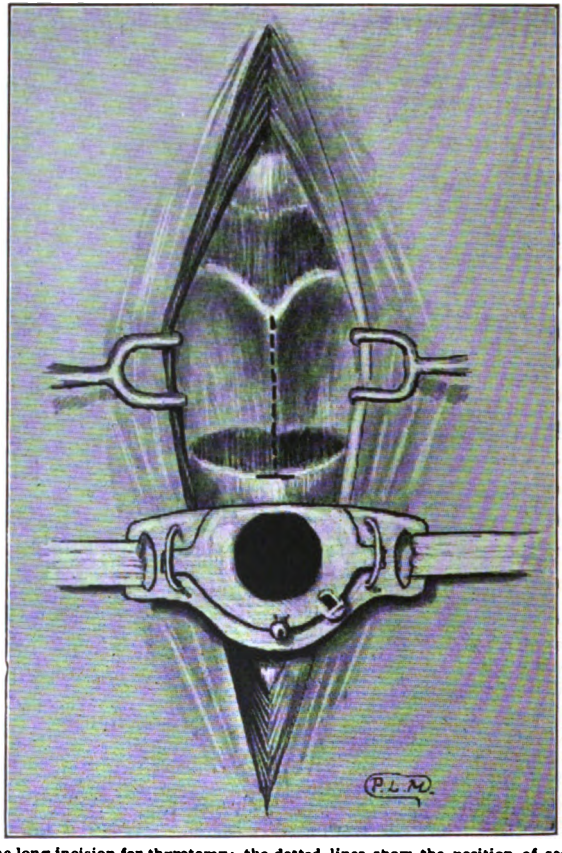

FIG. 1.—The long incision for thyrotomy; the dotted lines show the position of section of the cartilage; the tracheotomy tube is in position. (After Moure.)

will be surprised to find that it is always more extensive than he was led to anticipate from laryngeal examination. The decision is now arrived at as to the extent of the operation to be performed. Whether the growth is alone to be removed, the tissues at its base, or the lateral ala of the thyroid cartilage itself. The operation is

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usually done for what may be termed potential epitheliomata, warty growth with cellular infiltration at the base, in adults, or for actual epithelioma of one or other of the cords. The tissues from which the growth springs are generally freely removed. It is now my practice to paint the growth with a 20 per cent. solution of cocaine, to which is added 10 grains of suprarenal extract dissolved in half a drachm of cold water. The extract must be freshly made. The diminution of troublesome local oozing by this means is highly advantageous and striking. A small and carefully purified Turkey sponge is pressed into the trachea at the lower angle of the laryngeal wound. It should have a piece of silk attached. This is an additional preventative against blood passing down the air passages; I regard it as an invaluable aid, and shall relate a case where I relied upon it alone, an ordinary tube being inserted below. The growth is now grasped with "mouse-tooth" forceps, and freely cut away. I do not hesitate to sacrifice all the soft parts down to the cartilage beneath. Pressure is made on the wound inside the larynx with minute sponges held on forceps, and wrung out in spirits of turpentine. I next apply a fine and sharp curette, and finally the galvano-cautery at a cherry-red heat, sparing no pains to render extirpation as free and as extensive as possible. The application of the cautery finally checks all oozing. A little finely-powdered iodoform is dusted upon the part, and the thyroid cartilage is united with strips of kangaroo tendon. Here the use of a fine drill must be employed if the cartilages be markedly calcified.

The incision is accurately united with fishing gut and horsehair right down to the tracheotomy tube, and here a point of great importance has to be considered, the question of withdrawal or retention of the latter. As a rule, I now remove the tube so soon as ever the patient has recovered his "coughing reflex" and partial sensibility. Any blood which may then enter the air passages is readily expelled by coughing. The lower part of the wound, after the withdrawal of the tube, is not closed, but covered with a pad of iodoform gauze and kept carefully cleansed. The diagram (p. 202) shows the extent of the incision, the position of the suture in the thyroid cartilage, and the tracheal tube in position.

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Treatment after Operation.

The dressing of the neck is very simple, and mainly consists of collodion to the upper part of the wound and iodoform to the tracheal opening. About the fourth day the margins of the latter are approximated by adhesive strapping. The patient keeps the sitting posture in a "steam tent," and occasionally inhales the vapour of creosote. Three times a day a spray of "Listerine," mixed with equal parts of tepid water, is used for the larynx. No use of the voice is to be permitted. The patient must be provided with a slate to write his wishes and replies. I have found that patients swallow with surprising ease after this operation. I usually employ rectal alimentation for two days. I then commence to administer meat lozenges, which are gradually sucked. Thirst is relieved by small lumps of ice. At first swallowing is best performed by the patient lying upon his side, the tube of the feeder being placed in the dependent cheek. Should any slight fetor be noticed in the breath, and this is not normal on the third or fourth day, I add a little carbolic acid to the "Listerine" spray. In about 14 to 20 days convalescence is established; indeed, the recovery is very easy and quick. I never allow the voice to be used for a full month, and then only in a quiet whisper. The recovery of the vocal functions is ultimately quite surprising, though the intonation is always hoarse. The following cases may now be related :—

Case 1.—*Fracture of the Larnyx, Dyspnœa, Tracheotomy, Obstruction of the Glottis by Cicatricial Tissue, Thyrotomy and Removal; Complete Recovery.*—This case has already been reported very fully in the 'Lancet' for November 14th, 1896, but to make the series complete I reproduce its main features. A woman, aged 23, sustained a severe fracture of the larynx, which was asserted to have been caused by her walking in her sleep and falling over a chair. I always looked upon this statement with suspicion, and a belief that the injury was sustained from personal violence. She was admitted into St. George's Hospital on July 10th suffering from dangerous dyspnœa, and the house-surgeon, Dr. Fison (now of Salisbury), performed tracheotomy, and thus saved her life. She was discharged to all appearances well, on July 29th, the tube having been removed on July 20th. She had complete aphasia, but breathed well. On July 31st, however, an attack of dyspnœa came on. She returned to the hospital, where the wound was opened, and the tube again inserted. The case was watched through August, and the larynx gradually became stenosed. The cords were thickened, and beneath them some cicatricial formation could be plainly seen. In September thyrotomy was performed, and the larynx below the cords was nearly closed by a cicatricial web, which was continuous with a collar of indurated tissue and the cartilage externally. This was obviously due

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to chronic inflammatory action about the fracture. No projecting part of cartilage could be felt. The tissue was cut away with scalpel and forceps and the larynx strongly stretched open with the finger. The operation was done in the usual way, and there was nothing to note about the after-treatment. By August 23rd all the parts were healed and she was walking about the ward. She made a good recovery and was seen two months after. She breathed well and her intonation was a little hoarse, but surprisingly good, considering the extensive operation done. The cords remained greatly thickened.

CASE 2.—*Syphilitic Stenosis: Ultimate Result Unfavourable.*—A man, aged between 30 and 40, was admitted into St. George's Hospital in May, 1897, and had tracheotomy performed on account of sudden and urgent dyspnœa. He was suffering from manifestations of tertiary syphilis, and was placed upon full doses of iodides. The appearances with the laryngoscope were as follows:—Great swelling all round the aperture of the larynx; anteriorly a glimpse could be obtained of a growth, smooth on the surface, which seemed to originate very far down below the cords. Both cords moved imperfectly, and remained widely abducted, as though nerve lesion were present. After taking iodides for several months the swelling greatly reduced, and the formation could then clearly be seen; it was smaller than before, and uncertainty still existed as to its distinct nature. The cords were still widely abducted and motionless. On stopping the tracheotomy wound urgent dyspnœa was at once produced. Doubt occurred in this case as to whether the dyspnœa, especially marked during inspiration, was due to abductor paralysis or to the presence of a neoplasm, and accordingly it was decided to perform thyrotomy. This was done in June, 1897. There was a special difficulty about the operation, owing to the calcification of the thyroid cartilage, which had to be divided with fine cutting forceps. On opening the larynx no distinct growth was found; there was a swelling below the cords, consisting almost entirely of inflammatory thickening, covered with a prominent and rugous fold of mucous membrane. This was cut away with curved scissors. Recovery from the operation was uneventful, but no benefit was obtained, and the patient was discharged still wearing the tube. The wound at the site of operation persistently contracted, with the formation of much scar tissue. Under cocaine the larynx was thrice dilated, and once under anæsthetics the parts were stretched with the finger. The patient became intolerant of further proceedings, and preferred to wear his tube. When relating a series of cases of any operation, it is, perhaps, as important to relate the failures as the successes, and I should not be disposed to repeat this operation for syphilitic stenosis unless a very distinct neoplasm or "web" of tissue could be removed.

We next come upon a series of four cases where this operation was performed for growths of the larynx, with results only to be described as excellent.

CASE 3.—*Recurrent Papilloma; Thyrotomy and Excision of Growth; No Recurrence.*—I wish to relate this case very shortly, lest the identity of the patient be discovered. A cleric, aged 54, began to suffer in 1898 from dyspnœa and hoarse intonation. A growth was discovered on the left cord, and a portion of this was removed with forceps, and was stated to be papillomatous. It was then removed in three separate sittings by

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a skilled laryngoscopist, and after some applications of a caustic nature a cure was reported. The patient stated that he recovered his voice and breathing well. In three months fresh symptoms arose, and he went abroad, when so far as could be made out, similar operations were performed again with benefit. I saw him towards the end of 1893. He then had a growth on the left cord, which looked the size of a pea, and was of a pink colour. The larynx was swollen and inflamed, and a complete view of the growth difficult to obtain. He was hoarse and had some slight inspiratory dyspnœa; he was a stout "full-blooded" man. No induration or glands could be felt in the neck. I treated him with leeches and iodides for a fortnight, and then performed thyrotomy. The growth on exposure was, as usual, more extensive than could be estimated from above. It was the size of a cherry, sessile, and implicating both the cord and the side of the larynx. I cut it away freely, curetted the side of the larynx, and applied the galvano-cautery. The bleeding was very free, and I employed the manœuvre before spoken of—namely, plugging the trachea below the growth and above the tube with a small Turkey sponge. The only special remark I have to make about this case was the freedom of extirpation. The whole of the soft parts on the left side of the larynx were removed. Parts of the growth were sent to three good observers. One stated it was epitheliomatous, another papillomatous, and a third called it a degenerative papilloma, potentially cancerous. The fact was that the growth was inflamed, and its true characters difficult to determine. I informed the patient that the prognosis of his case was dubious. He made a rapid recovery, and the only after-anxiety was some bronchitis with fetor of the breath on the fourth day. He recovered his voice sufficiently to articulate hoarsely, but never to do his duty. He remained perfectly well for four years. I saw and examined him at intervals. He died in 1898 from "influenza and pneumonia," but there had not been any return of laryngeal disease. I purposely omit the names of the medical men who were concerned in this case, but I may state that the highest skill was brought to bear on the previous laryngeal efforts, and they were probably performed as well as it is possible to execute such operative proceedings by the way of the natural passages.

CASE 4.—*Fibroma: Thyrotomy: Removal by Galvano-Cautery: Recovery of Voice.*—A single woman, aged 33, came to St. George's Hospital in March, 1897. She had had good health until three years previously, when she gradually began to suffer from dyspnœa and loss of voice. The aphasia got worse and worse, so that she could only use her voice a little in the summer. She had occasional severe attacks of coughing with dyspnœa, which lately had been increasing, and had alarmed her.

On examination of the larynx, a pale, lobulated, rather sessile growth, the size of a large pea, could be seen to the right side and a little below the cords. It seemed to spring from the left cord, and narrowed the aperture of the larynx to a mere "chink." Dr. Whipham saw this case with me in consultation, and agreed as to the desirability of thyrotomy. The operation was performed on March 23rd. Dr. Dickson administered chloroform, and a preliminary tracheotomy was done. There was much venous oozing, and an enlarged layer of thyroid tissue lay in front of the trachea, which made the operation very difficult. A Trendelenburg's tube was inserted. The thyroid cartilage was split in the middle line, and pulled apart with sharp hooks. There was at this stage troublesome and profuse venous oozing, checked by pressure with a turpentine sponge.

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The growth, about the size of a large pea, now came into sight; it grew from the left cord. On being seized with the cold wire snare, its base was very tough and resistant, and it was found impossible to pull it away. Accordingly its base was slowly severed with the galvano-cautery. A tracheotomy tube was left in for a week, the parts being often sprayed with hydrogen peroxide solution. The tube was removed finally on April 1st, when the parts above were soundly healed. The final result of this case was excellent. In August, 1897, the patient had entirely regained her power of speech, which she had lost for six years. This was the more remarkable as great puckering and deformity occurred. The pathologists pronounced the tumour to be a fibroma and innocent in nature. It was thickly covered with a stratified epithelium.

The principal difficulties in this case were the profuse bleeding, which much obscured the parts. The Trendelenburg's tube acted well, no blood getting down into the bronchi. There can be no question that the bulk of this tumour could have been removed *per vias naturales*. There can equally be no question that its base could not be so dealt with. It was peculiarly broad, and to a large extent incorporated with the right vocal cord.

CASE 5.—*Removal of Large Papillomatous Growth from the Larynx, together with the "Cord" and all the Soft Parts.*—The patient was a Catholic ecclesiastic, aged 45. He came under my care in the summer of 1898. Hoarseness had gradually supervened for three years, and lately had progressed to almost complete aphonia. These symptoms had been much worse lately, and, in addition, "suffocation spasms" had recently been experienced. The case was under the care and inspection of Mr. Edward Blackett. The patient was a pale, ascetic-looking man. His voice was harsh and raucous, sometimes disappearing entirely. The left side of the larynx was occupied by a growth the size of a peach stone, which was lobulated, livid in colour, and seemed to have a broad base. There was no glandular enlargement in the neck. Under cocaine the mobility of the growth was freely examined with a curved probe, and it was found to be very sessile. Dr. Whipham also examined the case on several occasions, and the question of attempted extirpation *per vias naturales* was much discussed. Finally, thyrotomy was determined upon, and was performed on August 10th, 1898. There was nothing very special about the operation, excepting that the growth was very extensive; and I was uncertain as to whether the half of the larynx also should not be removed *in toto*. The bleeding was also profuse and troublesome. He made a good recovery without complications. Dr. Lazarus-Barlow kindly examined the growth, and found it to be of the usual nature—a warty papilloma, with abundant cell proliferation, but not truly epitheliomatous. For many months this gentleman's voice remained only a whisper, but his phonation gradually improved, and I have had a chance of examining him two years and four months after the operation. His voice is so surprisingly good that it is difficult to make observers believe he has had an operation done, and this is again borne out by the extraordinary changes in the larynx itself. The right cord is so mobile as to come over to the left side of the middle line, and here exists a ridge of mucous membrane or scar tissue, which looks at first sight exactly like a vocal cord. There

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is no sign of return of the tumour, but the aperture of the larynx is a little oblique or deformed. The result of this case has made a great impression on my mind as to the beneficial effects of this operation.*

The next case is equally satisfactory, though at the time of writing this paper only nine months have elapsed since the operation, so that one can hardly pronounce the certainty of a permanent cure, yet I believe such a result is exceedingly probable.†

CASE 6.—*Sessile Papilloma removed by Thyrotomy; Recovery, with Improved Voice.*—A gentleman, aged about 40, strong, plethoric, "thick set," and given to field sports, saw me in February, 1900. He had used his voice a great deal in hunting, and had noticed hoarseness and dyspnœa gradually creeping on for about three months. An examination was made with great difficulty. He was unable to open his mouth to the full extent, and any introduction of the mirror caused violent spasm and retching. The tongue could not be protruded. Feeling sure that some serious laryngeal trouble existed, much patience was exercised, and the patient practised opening his mouth and breathing. Ice was applied to the pharynx, and a 5 per cent. cocaine spray. At length with a small mirror I distinctly saw a growth. It was only a momentary glimpse, and, beyond the fact that it was on the left side, I was unable to study its characteristics. I therefore administered iodides in full doses, and repeated examinations of the throat were made. By the middle of March I was able to see the growth mass distinctly, and it had obviously not diminished in size. Moreover, the patient had had one severe attack of dyspnœa, which caused him to start up in bed with a sense of suffocation.

I afterwards learnt that during this time the patient had taken other opinions, both in London and the provinces. I need not enlarge on the treatments advised, as they were quite inapplicable to the case. I felt certain that personally I was quite unable to remove this growth by the natural passages, for even after weeks of patience and practice it could only be seen with great difficulty. By my request Mr. Butlin saw the patient with me in March. He recognised the difficulty of good inspection of the growth, but concurred with me in advising thyrotomy. The operation was done in the usual way, and no difficulty was experienced. As the neck was short, thick, and plethoric, I made the superficial incision unusually free, from the hyoid bone to the sternum, and the fierce venous bleeding, easily checked, showed how materially a too small opening would have delayed the future steps of the operation. After opening the larynx, I passed a small Turkey sponge on a

* The patient was present at the meeting, and read aloud with a good and distinct voice.

† Recently seen (February, 1902), quite well, voice much improved.

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piece of silk down the trachea for about an inch, and saturated the growth with a strong solution of cocaine and suprarenal extract. I was surprised at its size. I had estimated it at that of a pea. It was quite as large as a cherry, and very sessile. After extirpation the cautery and curette were vigorously applied, and on withdrawing the little sponge a small clot was brought up on its superior surface. The Hahn's tube was removed so soon as "coughing reflex" was restored, and the patient was convalescent in 14 days. The wound healed at once. He had no bad symptoms. Microscopically the growth was a papilloma, but there was much cellular infiltration at its base, and one skilled observer was confident it was cancerous. Recovery of voice is in this case confined to a loud whisper, but the patient is in the best of health.

CASE 7.—*Thyrotomy for Foreign Body (Bone).*—On December 28th, 1899, I was called to see a young man suffering from dyspnœa. His face was dusky and covered with sweat; his respirations distinctly laboured, with evident inspiratory stridor, and his voice reduced to a hoarse whisper. He stated that four days ago he had accidentally "swallowed" a large piece of bone, that it "stuck in his neck," and he brought up some bright blood. The difficulty of breathing and the pain in swallowing had apparently only been severe enough to frighten him in the last 24 hours. His condition was one of distress and danger.

Laryngoscopic examination was attended with the greatest difficulty. The parts were much swollen, having indeed an erysipelatous appearance. In the narrow "chink" between the swollen thyro-arytenoid folds a distinct glimpse was obtained of a piece of bone; it appeared like a spicule of osseous substances, stretched across the larynx antero-posteriorly. The difficulty of seeing it, and the spasmodic and dangerous dyspnœa of the patient, made, in my judgment, any attempt at removal from the mouth very unwise. Seeing the perilous and urgent nature of the case, I at once made arrangements for operation. Chloroform was carefully administered by Dr. Blumfeld. I exposed the whole front of the larynx and trachea to the root of the neck by a free median incision, which was rapidly deepened; the very free venous bleeding was readily checked by forcipressure, though so free an opening. Tracheotomy was performed, and a Hahn's tube was introduced. On opening the trachea a quantity of pus was ejected from the wound, showing that the foreign body was already doing grave local mischief. The thyroid was calcified, and I had to divide it with powerful curved scissors. On opening the larynx I could not see the bone, but by the finger it was detected lying firmly impacted below the cords, and extending downwards to the body of the cricoid. Apparently it was tilted on its edge. So firmly was the bone impacted, that it required considerable force to extract it with forceps, and this demonstrated how hopeless would have been any attempt to remove it from above. The piece of bone was of the extraordinary size and shape shown in the photograph. It is hard to believe that so large a foreign body could have passed the glottis, and yet the patient survived, and was moreover able to breathe. Silk sutures were passed and not tied, and the wound was tamponaded with iodoform gauze. The operation

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occupied about 15 minutes, being much expedited by the free median opening. The tube was removed in 12 hours, and the wound, covered by lymph, was drawn together by the sutures, which had been previously passed. Thymol inhalations were employed. The patient made a good recovery. His voice long remained "low and gruff," but ultimately it quite recovered, and was literally as strong as ever.

Side view; actual size.

Front view; actual size.

The next case is not one of thyrotomy proper, but may be termed suprathyroid pharyngotomy. It affords an excellent illustration of how a growth near the superior aspect of the larynx may be reached and dealt with through an external incision.

CASE 8.—*Suprathyroid Pharyngotomy.*—A widow, aged 57, came to the throat department at St. George's Hospital in July, 1897. She had total aphasia and some dyspnœa. The aphasia had come on about 18 months ago, and had gradually become worse. She was a stout woman, inclined to emphysema, with a short, thick neck. It was only after repeated examinations with the laryngoscope that I was able to clearly distinguish and define the position of a new growth. The neoplasm apparently sprang from the anterior surface of the thyroid, and extended a little way along the surface of the stalk of the epiglottis.

On July 5th the following operation was performed under chloroform, ably administered by Dr. Menzies:—A transverse incision was made in the thyrohyoid space, and another at right angles to it along the front of the thyroid cartilage. The anterior jugular veins were divided and tied, and the sternohyoid and thyrohyoid muscles were also divided. Here an error took place in the operation, and I found myself working above the hyoid bone instead of beneath it. This part of the operation was unexpectedly difficult, owing to the profuse venous oozing and depth of the neck. At length the thyrohyoid membrane was exposed and divided, and the epiglottis, after dividing the glosso-epiglottic ligament, was drawn out of the wound by a ligature passed through it with a sharp curved needle. By this means the larynx was drawn right out of the incision, and the growth readily came into view. It was more extensive than was thought, and some small, irregular tuberculated masses extended along deeply into the epiglottis. It was readily removed with a snare, and the curette and galvano-cautery were deeply applied to the site of origin on the epiglottis, so as utterly to destroy the tissues in immediate relation. The larynx was then replaced, and the wound sutured. The growth was a papillary epithelioma. To the naked eyes it appeared epitheliomatous.

The patient was very ill after the operation, and a severe attack of pneumonia came on a week after the operation. The wound discharged

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at one spot, and some fluid food passed out. For the first four days she was fed by nutrient enemata, but afterwards managed to swallow by lying on the side with the head hanging over the bed. By August 17th the wound had nearly healed, and she was breathing well and commencing to recover her voice. On November 19th the patient was well and strong, and had no dyspnœa. Her voice was "shaky" and uncertain, but she could well make herself understood. There was still some swelling about the arytenoid cartilages. There was no trace of return of the growth. I last saw her in 1899, two years after the operation. She was in good health, and could speak well. The epiglottis was depressed, but there was no return of the growth.

I feel sure that in these cases it is an error to suture the external wound immediately. Sepsis occurs from the pharynx, and it is wiser to stuff with iodoform gauze and merely pass the sutures, not tie them, until granulations and plastic lymph appear in the wound. I believe that this method is far safer to employ in all operations upon the neck where a communication has taken place with the gullet or pharynx.

CASE 9.—*Case of Laryngeal Cancer: Removal of one half the Larynx, with temporary great benefit.*—There are many remarkable features about this case, which I shall incidentally allude to in the relation of it. A night watchman, aged 60, had suffered from sore throat and hoarseness for about three months. He was a pale, thin man, and when he came to the hospital, in September, 1901, was suffering from dangerous and increasing dyspnœa. Examination was difficult, but a lobulated growth could plainly be seen filling the larynx and springing from the right side. It was ulcerating, and seemed to spread to the pharyngeal region behind. On September 7th chloroform was cautiously administered, and a low tracheotomy carefully performed. This relieved all danger of dyspnœa, and the larynx was repeatedly examined and the growth touched to estimate the possibility of removal. I may here point out that time is well spent in these difficult cases in repeated examinations, and the operation of tracheotomy ought never to be postponed, as is so often the case, until the dyspnœa is urgent and death actually impending. On September 23rd I performed thyrotomy. I stuffed the trachea above the tube with a small Turkey sponge attached to silk, and it acted perfectly. I found the growth very extensive, and removed the whole of the right ala of the thyroid with raspatory and scissors. I unfortunately found distinct signs that the pharynx was involved. The galvano-cautery was applied freely. Bleeding was very profuse. After operation the wound was tamponaded with gauze, and a fresh sponge was placed in the trachea. The tube was removed in 10 days. The patient could then breathe well. The result of the operation has been complete relief from dyspnœa, and the patient can articulate in a whisper, though half his larynx has been removed. Recurrence of growth in the pharynx I looked upon as certain.

I last saw the case in January, 1902. The growth was returning in the depths of the wound. Dyspnœa was entirely relieved. The main interest of this case is the great difficulty of estimating the

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actual extent of growth in advanced laryngeal cancer. I spent a fortnight over the study of this case, and concluded it was free of the pharynx, in which I was wrong. The case also affords a striking contrast to the others. It illustrates forcibly a clinical truth which all of us may lay to heart. That if an adult, especially a male, should suffer from abiding hoarseness and laryngeal irritation, no time should be lost in getting a careful laryngeal examination, so that if a growth exists it may be detected and dealt with in its earliest stages. The results, as shown by the cases detailed to-night, are as good as neglected cases are disastrous and unsatisfactory.

The CHAIRMAN (Dr. F. de Havilland Hall) said he felt he would be echoing the feelings of the Fellows by thanking Mr. Marmaduke Sheild for his admirable paper. He congratulated him on the success which had attended his operations, and thanked the gentleman who had been kind enough to show himself that evening. He (the Chairman) had examined the patient, and was of opinion that it was a most successful case, and the appearance of the larynx constituted a great triumph in surgery.

Mr. JONATHAN HUTCHINSON, jun., congratulated Mr. Sheild on his admirable results. In 1879 he was assisting the late Mr. James Adams, when that brilliant surgeon was at the London Hospital, on exactly such a case of intra-laryngeal tumour, in which the operation of thyrotomy was performed. The case was an admirable success, except that the man was subsequently hoarse. The growth was what would be called one in the pre-cancerous stage. He wished to thank Mr. Sheild for including a case in which he failed to relieve a syphilitic condition by operation. He might easily have left it out without anyone being the wiser. He had a case under care now in which he had done tracheotomy, and had been contemplating a second operation in the hope of clearing away cicatricial tissue. He would not touch it now after having heard Mr. Sheild's remarks. He concluded Mr. Sheild had mentioned the whole of his experience in unfavourable cases. It was interesting to find him alluding to the value of the local use of iodoform and turpentine to the wound, but he had a shrewd suspicion that the cases would do equally well without them. With regard to rubber versus sponge round the tube, he agreed fully with Mr. Sheild. He had seen the rubber bag come away in shreds. He asked whether, in the case of the woman who had pneumonia after pharyngotomy, tracheotomy was done. If not, would not that have saved the blood from getting into the trachea, and causing pneumonia? He also asked Mr. Sheild whether, supposing he had a case of definite cancer limited to a small part of the trachea, excision of one half would be sufficient? Also, where the glands were already affected, what would be the precise order of procedure? Would Mr. Sheild first remove the glands by a separate operation, and then do tracheotomy and excise half the larynx? Or would he do the whole in one operation? The question of an incision in a transverse direction being added to the median one, and the dissecting out of the glands would make little difference in point of time. One of the important lessons of the paper was that the cases, to be really successful, must be operated on in the pre-cancerous stage.

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Mr. WALTER G. SPENCER spoke in praise of Mr. Sheild's admirable series of cases. He would mention one or two points in which cases he had had differed slightly from Mr. Sheild's, although he (Mr. Spencer) had closely followed the lines laid down by Sir Felix Semon and Mr. Butlin. Mr. Sheild did not appear to have met with a case in which there was very difficult breathing, and the symptoms so urgent that as soon as anæsthetisation was commenced the tracheotomy tube had to be put in quickly. In some cases it was necessary to do laryngotomy by practically one stab into the crico-thyroid space, getting the tube in to relieve the patient for the moment, and then go on with the anæsthetic. With regard to the Trendelenburg tampon and the sponge, he did not think they had great advantages over an ordinary tube. Inconvenience occurred sometimes both with the rubber and with the sponge, which occasionally got dislocated. With regard to median thyrotomy, he preferred making a small incision either above or below the thyroid cartilage and aiding himself by a director inside, as in some cases if that were not done a cord on one side might be injured. Mr. Sheild's experience had been unusual in regard to the number of cases in which he had been able to unite the wound. In many of his own cases benefit had accrued by leaving the wound open, and by keeping the patient in an almost half prone position. Union was so rapid that much time was not gained by primary union. In an extensive operation on the larynx he thought it was valuable to leave the wound open, and then blow in iodoform, doing it at the moment the patient swallowed.

Mr. Sheild had mentioned that thyrotomy was not very successful in syphilitic cases. He (Mr. Spencer) showed a case before the Laryngological Society in which, three years after thyrotomy, breathing was easy, with only a hoarse voice. He believed that Mr. Sheild would have had a better result in his patient if he had cut out one of the vocal cords. He would have rendered the patient hoarse, but as long as the thyroid cartilage had not been destroyed, he thought success might be obtained by thyrotomy and the excision of a cord, and that most working men and some other people would prefer a hoarse voice to the wearing of a tracheotomy tube.

Mr. Sheild had not mentioned the other conditions for which thyrotomy was performed—namely, growths in children, and stenosis after tracheotomy for diphtheria. Those were troublesome cases. He had one case of chronic œdema of the arytenoid cartilages and false vocal cords cured by thyrotomy and excision. Thyrotomy might be done for fixation of the vocal cords from inflammation involving the thyro-arytenoid joints. Transverse pharyngotomy was shown by statistics to be one of the most unfortunate operations in surgery, because of the septic pneumonia, owing to wide division of the muscles and bleeding from the superior thyroid artery; whilst, in swallowing, food tended to be forced into the wound. Latterly he had been of opinion that median pharyngotomy, keeping strictly in the middle line, and extending even into the tongue, was more successful. A theoretical objection might be raised to the cutting through of the hyoid bone, but that was not a real difficulty. He felt sure that median pharyngotomy would be the more successful operation. He thought everyone would agree that Mr. Sheild was to be congratulated on his series of cases.

Dr. STCLAIR THOMSON said, though he had had no experience of supra-thyroid pharyngotomy, he felt there was a future for the treatment of growths above the entrance of the larynx, including those affecting the

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base of the tongue. He had seen several with surgeons, and one was alive 18 months afterwards without any sign of recurrence, the whole of the epiglottis and a good portion of the base of the tongue having been removed. In Mr. Sheild's seventh case the bone which was removed from the larynx was one of the largest foreign bodies which could be impacted there. He suggested, however, that if 12 or 24 hours had been allowed to elapse after the tracheotomy, the extreme irritation might have subsided, and then an attempt might have been made to remove the bone by the ordinary route. With regard to thyrotomies, he did not feel there was the field for that operation which Mr. Sheild appeared to anticipate. He had been on the look-out for such cases for some years, but had been struck by their infrequency. He had only had two private cases of the kind in four years, and one in hospital practice, though, in the latter work, he generally saw a hundred patients a week. The reason for the greater number in private practice was that educated people recognised the early onset of hoarseness, and were more particular about it. He thought the most satisfactory result in Mr. Sheild's cases was in that of stenosis from fracture. He thought Mr. Sheild's result in syphilitic stenosis was that of other experimenters. It was more useful in such cases to do a tracheotomy, and then to persevere with antisyphilitic remedies, including the inunction of mercury. He had known those remedies fail until after tracheotomy, and then the patient gradually recovered. The other cases of Mr. Sheild's appeared to have been those in which the malignant nature of the growth was not established: the third showed doubtful microscopical results, the fourth was a fibroma, the fifth a warty papilloma, and the sixth a true papilloma. He had not yet seen a simple growth of the larynx which he could not remove *per vias naturales*. When anæsthetics were given to children, papillomata could be removed, even without the laryngeal mirror. Cases which were difficult of examination were exceptional. He believed the future of thyrotomy was not a large one; its chief scope was in cases of malignant growths which were recognised early. He asked whether Mr. Sheild thought it necessary to drill the thyroid wings to bring them together. In the few cases which he (Dr. Thomson) had had, and others in which he had assisted colleagues, the portions of thyroid fell so naturally together that a few stitches through the perichondrium were sufficient to maintain them in natural apposition. Also, was the steam tent necessary? Many people were sceptical about that, because it kept out the fresh air, and was responsible for a chilling vapour on the bed. In cases in which there was apt to be a septic odour it was better to have more air and less tent. In three recent cases of thyrotomy for malignant disease much relief was felt from swallowing boiled water within the first 12 hours. One of his patients was out of bed in 24 hours, and in all recovery was rapid.

The CHAIRMAN (Dr. F. de Havilland Hall) said he cordially agreed with Dr. StClair Thomson in regard to the steam kettle and steam tent. He felt sure it was a great mistake in most cases. He had waged war against the poultice, and the steam kettle should be placed in the same category.

Mr. SHEILD, in reply, thanked the Fellows for the kind way in which they had received his narration of the cases. In regard to syphilitic stenosis, though operations were generally not happy in their results, he could conceive of cases of that condition in which benefit might result. He did not think any general rule should be laid down for application to all cases. Every case should be carefully studied and repeatedly examined

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before any operative measures were attempted. With regard to the insufflation of iodoform, there was much in Mr. Hutchinson's remark. He agreed it might be a bad application for putting into fresh wounds, especially extensive ones. Still, he believed that if applied to surfaces which were likely to suppurate, it helped to keep the parts clean, and to obviate fœtor. He used it where extensive curetting had been done, and, therefore, where there was likely to be sloughing. As to whether a preliminary tracheotomy would have prevented the pneumonia in one case, he did not think it needful if the external wound were not closed too early. He believed he erred in his anxiety to close the external wound. With regard to the question as to whether, in cases of cancer of the larynx in which the external glands were also involved, it was advisable to remove the glands at the same operation, he would say, as a general rule, that where the glands of the neck were involved the disease had progressed too far for successful extirpation. He had seen such cases operated upon, and had done them himself, removing the whole of the larynx, tying the carotid, and doing an enormous operation at the side of the neck. The immediate result seemed good, but, as a fact, it was a perilous operation, and recurrences were only too early. In all such cases the two operations should be done separately. The removal of the glands should be effected by turning up large triangular flaps, exposing the tissues freely, and doing a free dissection of all the glands in the sheath. Mr. Spencer spoke of median thyrotomy, which he (Mr. Sheild) had read of and proposed doing when he had a suitable case. Apropos of the closure of the external wound, he always sewed up the median incision, except the lower part into which the tube was inserted. The other part he drew together with strapping. Reference had been made to laryngeal papilloma and stenosis in children, and he had seen several cases operated upon. Of two cases one had thyrotomy twice and the other three times, yet both of them were as bad some time afterwards as at the start. That was in spite of curetting and the application of chromic acid with a freedom he would not care to emulate. He was aware of the treatment of snatching such growths out with the forceps, but the only case he had had was in private, in which tracheotomy was done. The growths ultimately disappeared spontaneously. In view of the difficulty these cases presented in children, he did not feel that it was the best method of treatment.

With regard to Dr. StClair Thomson's remarks, he thought that gentleman would agree that in no cases was the question of operation more difficult to determine than in those of malignant disease towards the base of the tongue and epiglottis. Probably the suggestion about the case of the impacted bone was right, but, as the bone was already causing ulceration inside the larynx, the risk of pneumonia would have been very great if something had not been done at once. He agreed that the cases needing thyrotomy were few in number, and he reminded Fellows that those he had quoted were culled from many years' private practice, and from large numbers of cases of all kinds in hospital.

Mr. Hutchinson raised a question which was the "crux" of the whole matter. In a man of 40 or 50 years of age, who was hoarse with a growth in the larynx, the difficulty of determining whether it was epithelioma or fibroma seemed to him very great indeed. Years ago he had taken such cases to laryngoscopists and skilled observers, and had got them to snip off a little piece of growth, which he afterwards sent to different pathologists of distinction. The reports on the specimens varied considerably, and led him to doubt whether the pathologists were able to identify them. The difficulty was not lessened by the fact that after a

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portion of growth had been taken away, and caustics or the cautery applied to the part, if there were anything epitheliomatous about it, there would be a quick recurrence. He agreed that the cases should be judged with very great care, and he did not suggest thyrotomy in all of them. The only growths of the larynx whch he had removed by the natural passages had been those which were pedunculated and moved freely about. These readily came away. But where the growth was sessile or broad, or where it was not in view, he did not see how thorough removal could be ensured without laying the larynx open.

With regard to drilling the thyroid cartilage in order to bring it together, he had notes of a case which was operated upon by another surgeon some years ago, without the thyroid having been sewn together. The parts slipped, and one half of the cartilage was at a lower level than the other, showing a very disfiguring ridge or displacement. Consequently, with this case in his mind, he always intended to bring the parts together in the way described.

In reference to the steam tent, he agreed it was a superstition, and perhaps they should all welcome its disappearance. But, seeing that even the College of Physicians still sanctioned the hanging of a carbolic sheet across the doorway in cases of scarlet fever, the steam tent should not be judged too severely.

He had had no experience of the operation for the removal of the epiglottis. Some years ago he saw two cases of malignant disease of the epiglottis, about which he had several consultations with other surgeons, but no operation was done, as they feared the after risk of food getting into the larynx. If he had another case he would operate upon it by the suprahyoid method.

CASES OF GASTROTOMY FOR RECENT GASTRIC ULCER.

By C. W. Mansell Moullin, F.R.C.S.

In two of these cases the immediate cause for operation was recurrent hæmatemesis.

The first case was that of a woman, aged 26, who had suffered severely from attacks of indigestion for the last 18 months. Of late the pain had been intense, coming on immediately after meals or within half an hour, and radiating through from the epigastrium to the back. Until the last week actual vomiting had only been of occasional occurrence. Since then it had been almost constant. There had been a slight attack of hæmatemesis four months ago, and other attacks becoming more severe seven days ago, five days ago (twice), four days ago (twice), and on the morning of admission. On the last occasion the quantity is said to have amounted to more than a pint. Shortly after admission she vomited 6 ounces of almost pure blood. There was no history of melæna,

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but an enema given the day she came in brought away a considerable quantity of blackened blood. The pulse was 140 to the minute, very small in volume, and of low tension, and the patient was decidedly blanched. My colleague, Dr. F. J. Smith, under whose care the patient had been admitted, thinking that it was not advisable that she should be allowed the chance of losing any more blood, very kindly had the case transferred to my wards, and an operation was performed as soon as it could be arranged. The abdomen was opened in the middle line, and the serous coat of the stomach carefully examined. As there was no scarring or other sign of inflammation, the stomach itself was packed round with sponges, and opened by a vertical incision midway between the pylorus and the cardiac end. The angles of the incision were held open by pressure-forceps, and the mucous surface systematically everted and examined bit by bit, that covering the posterior wall being brought into view by introducing the hand through an opening in the great omentum, and lifting the wall of the stomach into the wound. A large vaginal speculum, introduced through the opening in the anterior wall, enabled the cardiac orifice and that part of the mucous surface which could not be brought up to be inspected without difficulty. A small ulcer was found on the anterior surface near the greater curvature. It corresponded fairly well with the ordinary description of a simple ulcer, and had evidently been bleeding recently. There was no induration about its edges, and no vessel was visible on its floor. An incision was carried round the ulcer, and the whole of it excised down to the muscular coat, the gap that was left being closed by fine silk sutures, which served at the same time to stop the bleeding. The wound in the stomach was closed by a double set of sutures, and the opening in the abdominal wall secured with silkworm gut. Convalescence was uninterrupted, except for a slight attack of parotitis on the second and fourth days. The patient vomited once after the anæsthetic. Small quantities of hot water were given by the mouth the same evening, and continued at frequent intervals for the relief of thirst, but for the first four days the feeding was carried out entirely by means of nutrient enemata. Solid food (fish) was given 10 days after the operation, and a few days later the patient was placed on ordinary diet. She soon regained her strength, and since the operation has been perfectly free from the pain and indigestion which troubled her so much before.

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The second case was that of a married woman, aged 28, who was admitted into hospital on April 25th, 1900, for hæmatemesis. She was sent up in an ambulance with a history of having vomited blood at frequent intervals for four days. Shortly after admission she was sick, bringing up 15 ounces of dark-coloured blood, and became very much collapsed. An operation similar to that already described was performed as soon as could be arranged. When the stomach was exposed an old cicatrix was seen near the cardiac end. This was excised, and then a recent but chronic ulcer with indurated edges was found by its side. In the floor of this ulcer was a bleeding branch of the gastro-epiploic artery. The ulcer was included in the incision, and the opening in the stomach wall, which was nearly 3 inches long and ran almost up to the cardiac orifice, secured by a double set of sutures. The patient was very much collapsed, but rallied under injections of strychnia and caffein and rectal enemata. For the first three days vomiting was very troublesome. Then it ceased, and the patient appeared to rally. She was able to take small quantities of jelly by the mouth. The bowels acted at first after enemata, and then spontaneously. The tongue became moist. The abdomen was soft, not distended, and moved well with respiration. But, as in the former case, parotitis set in, first upon one side, then upon the other. Suppuration followed, and though relief was obtained when the abscess was opened, the patient died on the ninth day after operation. At the *post-mortem* examination, the opening in the stomach was merely held together by the sutures. The edges were sloughing, and there was an entire absence of repair. The kidneys were markedly granular and contracted. It is worthy of remark that parotitis occurred in both these cases, though there was suppuration in only one. As there had not been any case of post-operative parotitis in my wards for at least five years before, and there has been none since, it almost seems as if there must have been some connection between them; but they were not on the same floor, or in the same ward, and the one died 10 days before the other was operated upon, so that in all probability it was merely a coincidence, but it was a curious one.

In the third case, that of a patient who was under the care of Dr. Capon, the immediate cause was uncontrollable pain and vomiting. The patient was a married lady, aged 32. For some months she had suffered in a very severe degree from all the

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symptoms of gastric ulcer with the exception of hæmatemesis and melæna. These had never occurred. All the ordinary remedies, including prolonged rectal feeding, had been exhausted without obtaining the least permanent benefit. The patient had lost more than a third of her weight, and was so weak that she could scarcely raise herself from her bed. The pain came on within a few minutes after swallowing food, and was especially severe at the ensiform cartilage. The skin over this region was exceedingly sensitive. Vomiting shortly after food was the rule. This relieved the worst of the pain, but it usually continued with more or less severity for some hours after anything had been taken. After consultation with Dr. Maclagan it was decided to explore the stomach, and on opening it a long, narrow ulcer with slightly thickened edges was found lying at the bottom of one of the rugæ on the anterior surface close by the incision. It was about three-quarters of an inch in length, and extended down to, but not into, the muscular coat. No other lesion of any kind could be found, though the whole of the interior was carefully explored in the way already described. The ulcer was excised, the mucous and submucous layers sutured together, and the stomach and abdomen closed in the usual way. In this case also vomiting continued for some considerable time. The pain disappeared completely and finally, but owing to the sickness it was some time before the patient could take sufficient food to enable her to regain her strength. Recovery finally was complete.

I may mention that, in addition to these cases, I published in the 'Lancet,' of October, 1900, a series of three others, in which I had operated for hæmatemesis, all of which terminated successfully, the patients recovering completely without any drawback. Of five cases, therefore, in which I have operated directly for hæmatemesis, four have been cured, and I have little doubt myself that the fifth would also have survived had not her condition been so desperate.

It is usually held that the advisability of operating in these cases can be settled at once by comparing the actual mortality of hæmatemesis in cases of gastric ulcer, with that which it is supposed might follow gastrotomy performed under these conditions. The former is put down at from 3 per cent. to 5 per cent. The latter is estimated very variously, according to the kind and severity of the cases selected. No such comparison, however, is possible under the present conditions. On the one hand the whole number of cases of

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hæmatemesis, trivial and severe alike (and the trivial ones are much the more numerous), is taken, and the mortality percentage calculated from that. On the other, only those cases which are absolutely desperate, in which the operation is performed as a last chance, after everything else has been tried and has failed, are reckoned at all. No one suggests, or has ever suggested, operating upon slight cases of hæmatemesis. They should be placed entirely on one side and not taken into calculation. The only comparison that can be made with any pretence at fairness is between those cases in which surgical measures are advised and declined, and those in which they are advised and accepted. I have no doubt as to the result. In three of my five cases the patient was already so bloodless that transfusion had to be performed. In two others I had to decline to operate altogether—they were beyond even transfusion. The mortality of such cases, left to themselves without operation, is certainly not 3 per cent. or even 5 per cent.; it is much nearer 100 per cent. And they are the cases with which the only comparison can be made when the result of operation is the subject in dispute.

As no paper of this kind appears to be complete without statistics, and as the statistics of the mortality from gastric ulcer, and especially from hæmatemesis, vary a good deal according to the authority from which they are taken, I asked the medical registrar at the London Hospital, Dr. Wall, kindly to tabulate for me all the cases of hæmatemesis from gastric ulcer which had been admitted into the London Hospital in the five years 1895 to 1899 inclusive. All cases of hæmatemesis from cirrhosis, malignant disease, &c., were carefully excluded. The result is of decided interest. The total number was 246—202 women and 44 men; and taking one year with another the numbers were very much the same in each. The total number of deaths from hæmatemesis was 10, or 4 per cent. But the incidence of the deaths according to age was very singular. Under the age of 30 there were 153 women with only one death; over 30 there were 49 women with three deaths, or 6 per cent. Taking the men, there were only five under 30 years of age with one death; and 39 over 30 with five deaths. In other words, the mortality from hæmatemesis among the men over 30 years of age is 12½ per cent.; and taking all the men together, regardless of age, nearly 14 per cent. So far, then, as the question of operation for hæmatemesis is concerned, there must, it appears to me, be one rule

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for women under 30 years of age, and another for women over 30, and especially for men. But in considering the advisability of operating in general in cases of gastric ulcer, there is another point that must not be forgotten. These figures are only concerned with the mortality caused by hæmatemesis. There are other causes of death in gastric ulcer besides this, and those causes must also be taken into consideration. When the percentage of deaths due to these is added to that due to hæmatemesis it must be acknowledged that the death-rate due to gastric ulcer, especially in the case of men or of women over 30, is much higher than is usually believed.

I am fully aware that the treatment of gastric ulcer is, and must remain, in the hands of physicians. But it is becoming more and more evident every day that there are certain contingencies or accidents which may occur in the course of a case of gastric ulcer which, if the consequences are to be avoided, can only be met by the adoption of prompt and decisive surgical measures. This is admitted so far as perforation is concerned; but I maintain that the principle should be carried much further. It should include all those cases in which, in spite of rectal feeding and other remedies, the pain after food is taken continues with severity, as it did in the case I have described, in which vomiting after food obstinately persists, in which the patient is steadily losing ground, and in which medical treatment has been tried thoroughly for a sufficient length of time—say, for some months—and has failed.

So far as hæmatemesis is concerned, it is clear that age and sex make a very material difference, and much greater risk may be run in the case of women under 30 years of age than in the case of those over 30, and especially in the case of men. My own figures stand at only one death in 153 cases of hæmatemesis due to supposed gastric ulcer occurring in women under 30. (I may say that I have reason to believe that that particular period of five years was an exceptionally heavy one.) Surgical treatment, however, can rarely be required in them. But if there is one single severe hæmorrhage in a case in which the previous history suggests the presence of a chronic gastric ulcer (when the bleeding is probably due to an opening in an artery of some size, and is not merely capillary); if there are two separate attacks of severe hæmatemesis at a short interval, or if there are frequently such hæmorrhages, so that the patient is becoming seriously anæmic, I believe that the risk of operation is very decidedly less than the risk of leaving the patient

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alone, and that the best interests of the patient would be consulted by giving him or her the chance of operation before the condition becomes desperate.

The CHAIRMAN (Dr. F. de Havilland Hall) thanked Mr. Moullin for his most interesting and instructive communication. Curiously enough, though he always had two or three cases of gastric ulcer in his wards, he had not yet had a case in which it was necessary to call in a surgeon, either for severe and repeated hæmatemesis, or for perforation. Once or twice he had been doubtful about the best course, but, so far, the patients had recovered without operative interference, and he could not recall a death from hæmatemesis arising from gastric ulcer in his wards. Still, he was prepared to agree in Mr. Moullin's indications, and thought physicians would be culpably indifferent to the welfare of their patients if they neglected to seek the assistance of their surgical colleagues where such indications were present. He was particularly interested about the occurrence of parotitis in gastric ulcer, as he was at present seeing from time to time in consultation a case of what he believed to be duodenal ulcer, where convalescence had been interrupted by a severe attack of parotitis, which, however, was subsiding without suppuration. He understood that suppuration occurred in 50 per cent. of the cases of parotitis coming on in connection with abdominal operations. Professor Lebert, in his treatise on ulcers of the stomach, estimated that four-fifths of the cases of gastric ulcer, at some period of their course, had hæmatemesis. In severe cases of hæmatemesis he had been accustomed to keep the patient absolutely at rest, and to feed by suppositories for four or five days, and that he usually found sufficient. He took care to give nothing by the mouth except a little hot water, or a piece of ice to suck, whichever the patient preferred. He found it best to relieve the pain by hypodermic injection of morphia.

Dr. VOELCKER joined in congratulating Mr. Mansell Moullin on the success which had attended the majority of his cases. He thought physicians must recognise the fact that they ought to call in the aid of the surgeon in these cases more frequently than they did. About two years ago he saw a case which had a previous history of gastric ulcer, and he was sent for to the hospital because the patient had suddenly become worse. He found evidences of a perforated gastric ulcer—the liver dulness which had been present on first examination had disappeared. He saw the case with a surgical colleague, who opened the abdomen, but found no lesion on the outer side of the stomach. It was decided not to open the stomach, the abdominal wound was closed, and the patient made a very good recovery, and as there was no return of symptoms the patient left the hospital. Subsequently the patient had a severe attack of hæmatemesis, from which she nearly died. He felt no doubt that if the stomach had been opened at the operation the ulcer would have been found and dealt with, and the condition cured instead of the urgent symptoms being merely relieved. It appeared from Mr. Moullin's experience that one could, with a certain amount of impunity, open the stomach and explore it thoroughly. In the out-patient department one saw patients with gastric ulcer who came week after week, month after month, and even year after year. He had one patient, a woman, who had attended for over two years with a chronic gastric ulcer, who refused operative procedure, but he felt sure that treatment at the hands of a

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skilful surgeon would cure her. He thought the cases in which most good would be done by operation were those where pain was prominent. The mere arrest of hæmorrhage from a gastric ulcer might not be very difficult to attain by medical means; but he thought mere arrest of hæmorrhage sometimes led to more or less of a fool's paradise in regard to the eventual outcome. He asked whether Mr. Moullin experienced any difficulty in excising the scar of a chronic ulcer. When chronic ulcers were situated towards the pylorus he thought it must be a difficult matter to get away all the cicatricial tissue.

Mr. Mansell Moullin, in reply, congratulated the Chairman on his immunity from disaster in connection with gastric ulcer. The real difficulty was the separation of cases of capillary hæmorrhage from those of hæmorrhage from an ulcerated artery on the floor of an old gastric ulcer. If a branch of the splenic or gastro-epiploic artery opened out on the floor of the ulcer, no medical means could possibly stop the hæmorrhage. With regard to excision of the gastric ulcer, in his case there was no induration. Sometimes the induration was so great that it was impossible to excise the ulcer, and the only thing to do then was gastro-enterostomy. He could not help remarking on the singular novelty that an operation of this kind should be so blessed by physicians.

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March 24th, 1902.

ON SOME POINTS IN THE TREATMENT OF PUERPERAL ECLAMPSIA.

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THE points to which I wish to direct attention are two. First, what is the effect of emptying the uterus in eclampsia? Will it stop the fits? Does it do good? Secondly, the effect of the tepid bath used to reduce temperature in certain cases of eclampsia.

1. In the treatment of eclampsia it has been for many years said that it is important quickly to empty the uterus. One excellent and popular recent text-book says that eclampsia differs from the fits of scarlatinal nephritis, "inasmuch as they can be arrested at once by emptying the uterus." The author goes on to apply this principle, and he says that the first thing in treatment is "the induction of labour by the most rapid means consistent with safety." The practical precept follows naturally from the assertion made as to the clinical history of the disease. Another recent writer, after quoting the statement that "emptying the uterus is a certain means of checking the fits," says that he prefers "to modify the statement by saying that if the patient survives the emptying of the uterus she will almost certainly recover." Further on the same writer says: "The shorter the duration of labour, the better the prognosis of the case." Dührssen,* of Berlin, has devised an operation which he calls vaginal Cæsarean section, by which he says he can empty the uterus easily and safely in five minutes, and which he recommends as a sort of specific for eclampsia. Up to the date of publication of his latest paper he had only done the operation three times for eclampsia, although many cases have been treated in a way approximating to it.

This theory, that rapid delivery is beneficial, is no novelty, nor is it a new idea to cut open the soft parts in order that delivery may be quicker. Velpeau † in 1834 advocated the acceleration of delivery by incisions, and this was practised by Kiwisch, Scanzoni,

* 'Archiv für Gynäkologie,' Band lxi.

† 'Des Convulsions chez les Femmes,' Paris, 1834.

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Bodin, and others. In the work of Chiari, Braun, and Spaeth,* published in 1855, it is definitely stated that the artificial induction and acceleration of labour diminish the risks to mother and child arising from uræmic convulsions.

If these methods of treatment really produced the benefits expected from them, it is strange that the profession should have been so slow to accept them, and that more than half a century afterwards a German teacher should resuscitate the acceleration of delivery by incisions, and that German professors should think it necessary to analyse laboriously hundreds of cases to show that prognosis is improved if delivery is hastened. Real improvements in the treatment of disease do not take half a century to become accepted. No one at the present time would think it needful to collect statistics to prove that antiseptics prevent septic poisoning, that the removal of ovarian tumours is good treatment, or that salicylates will shorten the course of acute rheumatism; and yet these were things unknown when Velpeau wrote. After more than 50 years of trial it is thought that the profession still needs to be persuaded that hurrying delivery in eclampsia is good treatment. Possibly those who so teach may be themselves a little uneasy, and may have a sub-consciousness that the foundation of their faith in this matter is a little insecure and needs support. Hence the pains recently given to the subject.

Prognosis in puerperal eclampsia is exceedingly difficult. It is possible, though difficult, to recognise when a patient with this disease is moribund. If the patient is recovering, as evidenced by cessation of fits, less depth of coma, and re-establishment of the secretion of urine, it may be safe to give a favourable prognosis. But early in the case, when the patient has had one or even a few fits, and is in coma with almost suppressed urine, but the temperature is not very high or the pulse very rapid, it is not possible to say whether she will recover or die. This being the case, little confidence can be placed in conclusions as to the effect of treatment based upon a small number of cases. If three or four cases treated in a particular way recovered, before accepting their recovery as the result of treatment we must remember that about 80 per cent. of those suffering from the disease recover under the most different treatments, and that therefore the probability is that these three or four patients would have recovered under any treatment not in

* 'Klinik der Geburtshülfe und Gynäkologie,' Erlangen, 1855.

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itself harmful. The true test of the effect of treatment is the power of predicting its effect. In a case of eclampsia, in which the patient is not obviously either dying or recovering, we cannot yet predict what the effect of any remedy will be. The next best test is the comparison of large numbers of cases. If a treatment has cured three or four cases it will cure also 300 or 400 cases. I purpose to apply this test to the statement that when the uterus has been emptied the fits at once cease, and to the doctrine that the first thing in treatment is to empty the uterus.

Is it true that the fits cease when the uterus is emptied? Schauta * has analysed the records of the second lying-in clinic of Vienna from its foundation in 1834 up to the end of June, 1880. During that period there were 344 patients who suffered from convulsions. Two of these were considered to be cases of ordinary epilepsy and not eclampsia. There remain, therefore, 342 cases of eclampsia. Among these there were 42 in which the fits began during pregnancy, 185 in which they began during labour, and 82 in which the disease appeared after delivery. Now, of the 185 in which the fits began during labour, in how many was the disease "arrested at once" when the uterus was emptied? In 123 the fits continued—in 50 with increased severity, and in 38 with lessened severity. In only 62 the fits ceased with delivery. How can the statement that the disease is arrested at once by emptying the uterus be reconciled with these figures?

Schauta quotes a paper by Brummerstadt (and it is also quoted by Dührssen) containing records obtained at Rostock. I have not been able to see the original paper. He, out of 63 cases, found that the fits ceased after delivery only in 18, became less severe in 17, and continued unaltered in 28. Schauta remarks: "Without doubt this result is contradictory to the result stated in all text-books, that attacks beginning during labour cease with delivery or soon afterwards, so that, as a rule, delivery has a favourable influence on eclampsia. My cases show that the attacks in the great majority of cases do not cease with delivery, but in a number they continue with greater severity."

Schreiber † has continued the work of Schauta, and has analysed the cases observed in the second Vienna lying-in clinic from 1880, where Schauta left off, up to 1895. During that time there were 137 cases of eclampsia. In 23 of these the attacks began before

* 'Archiv für Gynäkologie,' Band xviii. † *Ibid.*, Band li.

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labour, and in 85 before delivery. Among the 23 in which the disease preceded labour the attacks did not occur after delivery in 16. In four they continued after delivery, and three patients died undelivered. One of the four in whom they continued after delivery was delivered by the quickest of all ways of emptying the uterus—viz., Cæsarean section. Among the 85 in which the disease supervened during labour, in 47 there were no attacks after delivery, and in 38, or 44·7 per cent., they continued.

Lantos* has analysed the records of the first obstetrical and gynæcological clinic at Budapest, under the direction of Professor von Kezmarszky, from 1863 to 1887. During that time there were 53 cases of eclampsia. In 39 of them the attacks began before delivery, and in five before labour. Of the five which began before labour none of the patients had fits after delivery, probably because the disease had had time to run its course. Of the 39 in whom the fits ought to have at once ceased after delivery, in 12, or 30·79 per cent., they continued.

Goldberg † has tabulated the cases occurring in the Dresden lying-in hospital during the time that it has been under the supervision of Professor Leopold, from September, 1883, up to June, 1891. In that time there were 81 cases. The notes of these cases have evidently been very imperfectly recorded, for in most there is no mention what the effect of delivery was. In 19 it is stated that after delivery there were no more fits; in eight fits continued after delivery. This very fragmentary record is of no value in showing the relative frequency of continuance or cessation of fits, but it is an additional lump of evidence to show that after delivery fits do not "at once cease."

Bidder ‡ has examined the records of the St. Petersburg lying-in hospital from January 1st, 1873, to December 31st, 1891. Within this period there occurred 455 cases of eclampsia. The records are imperfect, and therefore Bidder is not able to give so full an analysis of his cases as some of the other authors whom I have quoted have done. But he makes some statements which I extract. In 41 cases, in 38 of which the attacks began before labour, the patient had recovered before delivery. In 108, or 33 per cent., the fits ceased with delivery. Then, he says, "comes a series of cases in which

* 'Archiv für Gynäkologie,' Band xxxii.

† *Ibid.*, Band xli and xlii.

‡ *Ibid.*, Band xliv.

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one or a few slight attacks occurred after the birth of the child, and in which therefore the favourable effect of delivery was strikingly proved. The number of these is hardly less than that of the first series." The imperfection of the records prevents exact figures from being given. This appears to me to be an example of the fallacious reasoning so often followed by writers on the subject. They forget that four-fifths of the cases of eclampsia end in recovery within a day or two. If the disease begins before labour, fits will generally cease before delivery; if it begins early in the first stage of labour, the last fit may be before delivery, or soon after delivery. The fact that recovery follows delivery does not prove that it is the result of delivery. Bidder's cases include 66 forceps deliveries after which fits ceased, and 40 after which they did not cease. After this he goes on to observe, very correctly, although not very consistently with his former reasoning, that if we knew that emptying the uterus would cut short eclampsia Cæsarean section would be good treatment. But he says: "We unfortunately do not know this. We see in a large number of cases attacks that have come on during labour, continue after delivery, and increase in severity, and even lead to death by coma without further attacks after delivery."

One of the most energetic champions of operative delivery in eclampsia is Dührssen* of Berlin. He has gone through the records of the Charité Hospital in that city from January, 1880, to March, 1892, and has tabulated all the cases of eclampsia, 200 in number. Here also the records are not in every case complete. The figures which bear on the effect of delivery on the disease are as follows. In 65 cases the eclampsia ceased with the emptying of the uterus; in 53 the fits continued after delivery—a rather voluminous exception to the alleged rule that after delivery the fits at once cease. In 40 of the 53 the disease improved after delivery; in 13 it got worse.

Zweifel† is an ardent partisan of the treatment introduced by Dührssen—that is, rapid delivery by cutting open the genital passage, instead of waiting for it to dilate and quickly pulling out the child. Is it the fact that when this is done the fits at once cease? The attacks ceased in 55 per cent.; in 45 per cent. they did not. Glockner‡ has continued Zweifel's work, and has analysed the records of the Royal University Women's Clinic at Leipsic from

* 'Archiv für Gynäkologie,' Band xlii.

† 'Centralblatt für Gynäkologie,' 1895, No. 46, *et seq.*

‡ 'Archiv für Gynäkologie,' Band lxiii.

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October, 1895, to October, 1900. Within this time 143 cases of eclampsia were observed. What do they teach as to the effect of delivery in checking the fits? The fits ceased after delivery only in 34 per cent. of the cases. But in three cases it was because the patient died; if these are deducted it comes down to 32·4 per cent. The fits continued after delivery in 66 per cent.

Olshausen* has analysed 200 cases which occurred between September, 1885, and April, 1891, in the Royal University Women's Clinic of Berlin. His figures are not given in full detail. Ten patients died undelivered, 111 were delivered spontaneously, and in 77 delivery was artificially hastened. The effect on the fits is stated in only 143 cases. The fits ceased in 92, and continued in 51. Olshausen assumes that in every case in which the fits either did not recur after delivery, or became less severe, the recovery was due to delivery. It seems not to have occurred to him that recovery might have been simply the natural course of the disease. Goedecke† has continued the work of Olshausen, and has analysed 403 cases of eclampsia observed in the Berlin clinic from 1892 to 1899. There were 268 of these, which showed the presumed influence of delivery upon the fits. In 160 of them there were no fits after delivery; in 108 the fits continued, or 40·3 per cent.

Wieger, whose original paper I have not been able to see, but whose figures are quoted by Dührssen, found that in 111 cases the fits ceased after delivery in 39, became less severe in 35, and continued, or got worse, in 37.

Löhlein has analysed 325 cases. I have not been able to see his original paper, so I know not where the cases were observed. I quote from a summary of it given by Dührssen.‡ In 249 the fits began either before or during labour. According to Dührssen the fits either ceased or improved in 80 per cent. As Dührssen, in every case in which there was any improvement after delivery, gives credit for the improvement to the fact that the uterus had been emptied, this means that in 20 per cent. the patient was either no better after delivery or got worse.

Auvard§ gives statistics from the Paris Maternité from 1850 to 1886. In 179 cases the attacks ceased after delivery in 60 and

* 'Volkmann's Sammlung,' Neue Folge, No. 39.

† 'Zeitschrift für Geburtshülfe und Gynäkologie,' Band xlv.

‡ 'Archiv für Gynäkologie,' Band xlii.

§ 'Eclampsie Puerpérale,' Paris, 1889.

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diminished in 31, while in 88 delivery had no appreciable effect upon them.

Stroganoff* has published a series of 58 cases without a death. He candidly admits that in previous years there had been a heavy mortality, so that this only represents a fortuitous succession of a number of mild cases. But they illustrate the effect of delivery. He says that in 50 or 60 per cent. the fits cease after delivery.

During the 10 years, 1891–1901, 38 pregnant women who were suffering from epileptiform convulsions were admitted into the London Hospital. Of the 38, 20 recovered and 18 died. This is a very large mortality, but it is partly due to the tendency of general practitioners to send to the hospital those cases that seem likely to end unfavourably. Thus two of the patients were admitted in deep coma, and died a few hours after admission. In two others there was reason to believe that the fits were due to cerebral tumours, and the cases were therefore not puerperal eclampsia in the proper sense. In one case there was no albumen, and there was double optic neuritis. In the other the diagnosis of cerebral tumour had been made two years previously by Hughlings Jackson; but as the symptoms had improved the diagnosis had been thought incorrect. The patient had optic neuritis; there was only a trace of albumen in the urine, which contained 2 per cent. of urea. In neither case was there a *post-mortem* examination, so that the diagnosis remained unconfirmed, but I think neither can be regarded as eclampsia. That the tendency is to send bad cases to the hospital is shown by the statistics of the Glasgow Maternity Hospital during the last 15 years, collected by Dr. Munro Kerr, which show a mortality of 47 per cent. Dr. Jardine, from whose paper † I quote them, says: "The majority of the patients are sent in after they have been having convulsions for many hours. It is rare for us to get them within 12 hours of the first fit." I have notes of the relation of the fits to delivery in 23 cases. In 18 there were no fits after delivery; in five the fits continued. Of the 18, in seven the fits had ceased long before delivery. In one cessation of the fits followed a dose of morphia; in another the patient was comatose, and died in coma without more fits shortly after delivery.

The figures I have quoted will be, perhaps, more easily grasped if I put them together in a tabular form. (*See* Table I.)

* 'Monatsschrift für Geburtshülfe und Gynäkologie,' Band xii.

† 'The Lancet,' June 15th, 1901, p. 1682.

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TABLE I.—*Showing the Effect of Delivery on the Fits.*

Authority.	Number of cases.	Fits ceased after delivery.	Fits continued after delivery.
Schauta	185	62	123
Brummerstadt	63	18	45
Schreiber	105	63	42
Lantos	44	32	12
Goldberg	81	19	8
Bidder	455	108 (33 per cent.)	—
,, (forceps cases)	—	66	40
Dührssen	200	65	53
Zweifel	129	55 per cent.	45 per cent.
Glockner	141	48 (34 per cent.)	93 (66 per cent.)
Olshausen	143	92	51
Wieger	111	39	72
Auvard	179	60	119
Goedecke	268	160	108
Herman	38	18	5

These numerical statements are records of a very large number of cases. Different cases of eclampsia differ much from one another as to the time at which the disease comes on, the number of the fits, and the duration of the attack. Whether or not they cease after delivery depends upon these conditions. In small groups of cases there is often an accidental preponderance of cases of a particular kind, and therefore it is that in some of the cases which I have quoted there is a large proportion, and in others a small proportion, of cases in which the fits persisted or ceased after delivery. But, looking at them broadly, and seeing in how large a number fits continued after delivery, it seems to me that the statement that *when the uterus is emptied the fits at once cease*, is one which it is impossible to reconcile with the facts. When the uterus is emptied the fits do *not* at once cease; emptying the uterus is *not* a certain means of checking the fits; when the uterus is emptied the fits often continue—in most cases they get better, in some they get worse. The natural history of the disease should lead us to expect, unless delivery were actually injurious, that 80 per cent. would get better.

It may be said, even if the contention that after delivery the fits at once cease be given up, yet it is the fact that speedy delivery is good treatment; that cases treated by accelerating

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delivery do better than those in which labour is allowed to take its natural course. Let us see if this is so.

Goldberg, in the papers which I have already referred to, gives the result in his 81 cases. The death-rate among the whole was 24·7 per cent. Among those patients delivered by operation it was 34·21 per cent. Among those naturally delivered it was 16·28 per cent.

The whole mortality in Dührssen's cases was 21 per cent. The mortality among those in whom the disease began before delivery was 23·7 per cent. Dührssen thinks that three of the deaths occurred from sepsis, and therefore ought to be deducted. If this be allowed, it brings the mortality down to 19·8 per cent. for the whole number, and 22·7 per cent. for those in which the disease began before delivery. In 80 of Dührssen's cases delivery was artificially accelerated, and in these the mortality was 23·75 per cent. In 30 delivery was allowed to take place spontaneously, and among them the mortality was 21 per cent.

The total mortality in Lantos's cases was 28·3 per cent. Of the 44 in which the disease came on before delivery, in 33 delivery was accelerated, and in 11 it was left to nature. Of the former cases 11 patients died, and of the latter two died. In other words, emptying the uterus was followed by 33 per cent. of deaths; letting it alone by 18 per cent.

The total mortality in the cases collected by Schreiber was 19·7 per cent. The mortality among those delivered by operation was 19·23 per cent., and among those spontaneously delivered 20·3 per cent.—an insignificant difference.

Zweifel writes as one convinced of the good results of Dührssen's practice of delivering by extensive incisions into the cervix and vagina. He tells us that the cases treated by him fall naturally into two groups: one from April, 1877, to the beginning of 1892, during which the principle of treatment was expectant; and the second from the beginning of 1892 onwards, in which Dührssen's practice was followed. The first set of cases numbered 49, with 16 deaths, and the second 80, with 12 deaths—a death-rate of 32·6 per cent. by expectant treatment, against 15 per cent. by active treatment. This looks convincing, until one examines the records of the actual cases. I then find that in the first, or the period of expectant treatment, many patients were treated by rapid delivery, either by forceps, turning, or craniotomy; and that in the figures are included patients who died undelivered, and cases in which the

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convulsions began after delivery. I find also that in the second period, or that of active treatment, not all were delivered rapidly —that there were patients who died undelivered, and whose fits began after delivery. And in both sections there are a number as to whom there is no record how they were delivered. The latter are numerous enough to prevent any numerical statement of the results of different modes of delivery. One patient was delivered by the most rapid of all methods—Cæsarean section—and had 30 fits after delivery. Zweifel remarks that the preparation of his statistical account of his cases has shown him how difficult it is to prove things by figures, and how difficult it is to guard oneself from false conclusions. Even if one is careful to keep in memory the impression derived from individual cases, yet one finds the impressions of experience greatly upset by numerical test.

The total mortality among the cases analysed by Glockner, after deducting that of one patient who was brought in dead, and that of another who died a few minutes after admission, was 17·24 per cent. But Glockner says that there were three others who were moribund when admitted, and these should be deducted; the mortality is then reduced to 15·49 per cent. 114 of his patients were delivered by operation, and the mortality among these was 17·5 per cent. Only 10 cases were left to nature, and three of the patients died, a mortality of 33 per cent. But I presume that the moribund patients were not delivered by operation, and only two died undelivered; so, if we deduct the third moribund patient, the mortality comes down to 20 per cent., nearly the same as that of the operative delivery. In some patients dilatation of the soft parts as a preliminary to operative delivery was not practised, I presume because it was not required. In some dilatation was effected slowly by dilating bags; in some rapidly by incisions, as recommended by Dührssen; and in others by the combination of incisions and dilatation. The object of the incisions is to empty the uterus as quickly as possible. Incisions were used in 46 cases, with 12 deaths, or 26 per cent. They were not used in 48; and in these there were only eight deaths, or 16·6 per cent.

In Olshausen's collection of cases the difference between the results of natural and accelerated delivery is so slight as to be practically nothing. In 111 natural deliveries there were 24 deaths, or 21·6 per cent. In 77 operative deliveries there were 16 deaths, or 20·8 per cent.

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Among Goedecke's cases there were 192 in which delivery was accelerated by operation. The mortality among these was 16·1 per cent. The mortality among the whole 403 cases was 16·9 per cent.

G. Veit * has tabulated 802 cases collected from different maternities. I use his figures for convenience, although they include some that I have already quoted. Of the 802 patients 446 were delivered by operation of some sort, with 114 deaths, a mortality of 25·5 per cent. 356 were not delivered by operation, and of them 74 died, or 20·8 per cent. I will put these figures together in a tabular form, so that they may be more easily seen :—

Authority.	Natural delivery.	Accelerated delivery.
Goldberg	16·28 per cent.	34·21 per cent.
Dührssen	21·0 „	23·75 „
Lantos	18·0 „	33·0 „
Screiber	20·3 „	19·23 „
Glockner	20·0 „	17·5 „
Oishausen	21·6 „	20·8 „
Schauta	26·0 „	43·0 „
G. Veit	20·0 „	25·5 „

It may be said—and I think the objection a fair one—that the figures showing a very high mortality among the patients delivered by operation are taken from pre-antiseptic times, and apply not to operative delivery under present-day conditions. This may be so; and I grant that operative delivery, performed skilfully under anæsthesia and with antiseptic care, is not likely to prove fatal, to aggravate the disease, or seriously to injure the patient. But if it were the fact that "delivery by the most rapid means consistent with safety" was the first, the most important, thing in treatment, then every accoucheur and midwife ought to do his or her best to hurry on delivery, no matter what the skill or experience of the operator, what the surroundings of the patient, or what the amount and kind of assistance at hand; and if this were acted on, I think that in some places operative delivery would have a higher mortality than delivery left to nature. I take the very best results of operative delivery as compared with leaving delivery to nature, and what do they come to? A difference in the mortality of 1 or 2 per cent. only. What surgeon would recommend an operation if all he could

* 'Comptes Rendus du Congrès International de Gynécologie et d'Obstétrique,' Genève, 1897.

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say in its favour was that his patient's chance of recovery was about 2 per cent. better if he had the operation done than if he were let alone? I submit that immediate delivery is *not* the first thing in treatment; that if it is beneficial the benefit is of the most trifling kind, so small as to be doubtful. The practice is one based upon theory, not upon experience. The advantage is so small that if hurried delivery were indiscriminately practised by all who attend labour, in all circumstances, the mortality arising from operative delivery would soon overbalance the trifling and doubtful benefit of emptying the uterus. I therefore think that *the teaching that "to empty the uterus quickly is the first thing in treatment" is bad*, because likely to lead to bad results.

If rapid delivery were the one thing needful in eclampsia—if it were the fact that emptying the uterus at once caused the fits to cease—Cæsarean section would unquestionably be the proper treatment. No other method of delivery is so prompt. Rupturing the membranes and dilating the cervix with tents and water-bags are procrastination in comparison with it. Those who have hurried delivery by the incisions into the cervix and vagina recommended by Dührssen have generally not made them until the cervical canal has been opened up. If the internal os has not been dilated it appears to have been the practice to open it up with tents or dilators, and then to hasten delivery by incisions. This is a far slower proceeding than Cæsarean section. It may be urged that if Dührssen's principles are applied to the full, and the lower segment of the uterus and vagina are cut open enough to allow the extraction of the child without waiting for dilatation, then delivery by the natural road may be accomplished as quickly as Cæsarean section. Possibly it may, but as yet it is not certain that vaginal Cæsarean section is less dangerous than the old Cæsarean section. Zweifel gives as a reason for preferring Dührssen's incisions that although Cæsarean section is a quicker method of delivery, yet the preparations take time. I see not why the antiseptic preparation of operator, instruments, and patient should take longer for one operation than the other. Döderlein gives as a reason against Dührssen's treatment that it is such a bloody and formidable thing that it cannot be done in private practice.

What are the facts as to Cæsarean section? Döderlein* has collected reports of 19 cases in which patients suffering from eclampsia were delivered by Cæsarean section. Of these 11

* 'Münchener Medicinische Wochenschrift,' 1894, No. 26.

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recovered and eight died. In 14 there were no more fits after delivery; in five the fits continued. In two of the cases the reason why there were no more fits was that the patients were moribund when the operation was done, and died shortly afterwards; one of these had had no fit for 12 hours before the operation. It is to be noted, also, that eight of the patients whose fits ceased, and four of those in whom they continued, had been treated by morphia. There are to be added to these the since published cases of Schreiber, in which the fits continued, and of Zweifel, in which there were 30 fits after delivery. Here we have the effect of the most rapid possible delivery, 12 cases in which it is possible to think that rapid delivery did good and seven in which it clearly did not.

Hillmann * has collected the records of Cæsarean section for eclampsia down to a more recent date. He finds 39 cases with 20 deaths. Of 41 children 18 died and 23 survived. In eight cases fits occurred after Cæsarean section.† I think such a proportion as this is far from decisive evidence in favour of the good effect of delivery. I may also remark that Cæsarean section generally involves free bleeding, which our forefathers held was the sheet-anchor of treatment in eclampsia.

The result of the foregoing part of my paper has been purely negative. If I have been successful in my contention, I have shown reason for thinking that a method of treatment widely recommended is not beneficial, and certainly not so beneficial as its advocates say. I now come to a more pleasing topic. I have a case to relate in which the prognosis appeared to be very bad, but the patient nevertheless recovered, and there was reason to think that treatment may have had something to do with her recovery. It is well known that some cases of eclampsia die with a steadily rising temperature. It is, I think, the experience of most that a very high temperature and a very quick pulse are of unfavourable import in the prognosis of eclampsia. If a patient is dying with a rapidly rising temperature, it seems to me rational treatment to try to bring down that temperature. I believe it to be the opinion of general physicians that the most powerful and most safe way of lowering the heat of the body is to abstract heat

* 'Monatsschrift für Geburtshülfe und Gynäkologie,' Band x, 1899.

† Hillmann says 40, but he includes one *post-mortem* section. He says that in seven the fits continued, but Zweifel's case is the seventh, and another is later in date.

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by the cold or tepid bath. The use of baths in renal disease generally, in the renal disease of pregnancy, and in eclampsia, as a means of promoting diaphoresis, is no novelty. It is a practice that can do little harm, and may do good. But this I am not going to discuss. I report a case in which the tepid bath was used for the purpose of bringing down temperature, and in which it did bring down the temperature, and it may have been the agent which brought about recovery.

The patient was admitted into the London Hospital on April 6th, 1901. Her age was 20 years. She had never been ill before, and was in the seventh month of her first pregnancy. She had sent for her medical attendant, Mr. S. R. Nicholls, at 12.30 a.m., because she had abdominal pain and vomiting. The bowels were confined, the temperature was normal, the patient had had no fit, and there was no vaginal discharge. A purge was prescribed. The medical attendant was summoned again at 4 a.m., because the patient's mother thought that she had had a fit, but the patient was quiet when seen. A simple enema was advised. At 8 a.m. the patient had an epileptiform fit. There was then no vaginal discharge, the temperature was normal, and the bowels had acted. A large injection of saline fluid into the rectum was ordered. Fits recurred, at first about once in every hour, then more frequently. At 8.30 p.m. the patient was admitted into the hospital (the notes of the case were taken by Dr. F. P. T. Hilliard, resident accoucheur, and Mr. G. S. Ward, clinical clerk). A hypodermic injection of a quarter of a grain of morphia was given at once. Soon after the patient had a fit, and another injection of a quarter of a grain of morphia was given. The patient was comatose. The temperature was 104·8° F., and the pulse was 174. The pupils were small. There was slight œdema of the legs. About an ounce of urine was withdrawn, and was found to be almost solid with albumen. The fœtal heart was not heard. The patient was put in a warm bath, which was rapidly cooled down to about 80°. She was kept in it for about 25 minutes, by which time her temperature had come down to 102°. The pulse became of lower tension. After being taken out of the bath the patient was wrapped in flannels, and was allowed to perspire. At 11 p.m. she had a slight fit, and soon afterwards she aborted. The fœtus was normal in appearance, not decomposing. On the 7th the temperature was normal. During the 8th and 9th it rose

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slightly, reaching 101° on the latter day. Next day it fell, and was not afterwards above normal. She was very restless, and her mental condition was not good. By the 20th she was so violent that she had to be removed to the workhouse infirmary, and thence she was taken to the County Asylum at Brentford. Owing to the patient's condition it was very difficult to examine thoroughly the retina, but there were no hæmorrhages in the parts seen. When admitted urine appeared to be passed unconsciously. About an ounce was withdrawn by catheter, which was almost solid with albumen. On the 8th 12 ounces of urine were collected, but the bowels acted four times, and some urine was lost with the stools. It was thick, with a deposit of urates. The albumen was almost entirely paraglobulin. The specific gravity of the urine was 1028; it contained 3 per cent. of urea. On the 9th, 58 ounces of urine of specific gravity 1024 were collected. There was a deposit of urates. It contained 3·4 per cent. of urea, and about one-eighth of albumen, about half of which was paraglobulin. By the 16th the albumen had diminished to one-twentieth, and it contained only a trace of paraglobulin. On May 26th, Mr. E. Vallance (medical officer to the West Ham Workhouse Infirmary) reported the urine to be free from albumen.

One case proves nothing. But I think that this was one in which anyone who ventured on a prognosis would have given an unfavourable one. I have seen one other case which has led me to think this practice a good one, but it occurred in private practice, and therefore I have no detailed notes about it. The patient during the lying-in period suffered from almost complete suppression of urine—about 1½ ounces were drawn off after she had passed none for 24 hours—which was solid with albumen. There were stupor and some muscular twitchings, but no actual fit, and the temperature was 105° F. I advised that the tepid bath should be used to reduce the temperature. This was done, the temperature was reduced, the symptoms were at once much relieved, and the patient ultimately recovered.

Dr. Amand Routh said the paper was a very valuable one, and Dr. Herman's work on eclampsia was well known by obstetricians. It was surprising, on looking at the statistics, to see how the growth in the belief of emptying the uterus more or less rapidly for cases of puerperal eclampsia did not really bear out its reputation. Until the present statistics were seen, one had no idea that the results were not much more favourable than they appeared to be. One of the reasons given was that the emptying of the uterus, or the steps preparatory to that procedure,

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did a great deal to relieve tension, not only the tension of the uterus, but the intra-abdominal tension. And one could quite understand why that should, in the hyperæsthesic state of the nervous system, help to remedy the nerve storm. In addition to that, there is a theory which seems fairly conclusive, that the eclampsia and the nephritis are really due to some poison in the blood, which was derived from altered metabolism of the fœtus or placenta. The idea was that if that interchange of material could be arrested by an expulsion of the fœtus and the afterbirth, the poison would cease to get into the maternal system. Those two reasons had led to the belief that the treatment was probably a good one. At the same time, he was not aware that any one in this country had advocated the extremely rapid method of Dührssen. The view generally taught was that in the first stage of labour the proper course was to rupture the membranes to relieve the tension. Then, when the head was on the perineum, by all means deliver the child by forceps. He did not know previously that rapid delivery was advocated quite so strongly as Dr. Herman had indicated.

With regard to the second point, tepid bathing in cases of hyperpyrexia, there was no doubt that was the proper method, because one could not combat the chemical changes proceeding in the system by any other means known at present. But the bathing was not always as successful as in the case Dr. Herman had related in his paper. He (Dr. Routh) remembered one case in 1882 which left a great impression upon his mind, and he had never seen quite such a bad case since. It was that of an unmarried girl who was having her first child. She was seen in the first stage of labour, being then apparently perfectly well. A few hours afterwards a doctor was sent for as she had had a convulsion. He (Dr. Routh) saw her soon afterwards. Her urine was drawn off, amounting only to a drachm or two, but it was almost pure blood, and became solid on boiling. She had one fit after another, and then became completely comatose. The labour pains were strong, and there was no necessity to do anything from the child's point of view. But the temperature steadily ran up to 107° in from three to four hours. She was placed into tepid water, in which pieces of ice were floated, and on several occasions within the next few hours her temperature was brought down to about 100° or 101°. But the water could not be kept cold enough. When she was taken out and put to bed, and an attempt made to encourage perspiration, the temperature ran up again almost immediately, and, in spite of all that could be done, she died from exhaustion plus hyperpyrexia. He remembered seeing the body about an hour after death, and even then the temperature under the arm was 109°, showing the enormous amount of chemical change which must have been proceeding. He was sure all the Fellows owed a deep debt of gratitude to Dr. Herman for nailing the method of rapidly emptying the uterus to the counter. Fortunately there were many other methods of proved value to fall back upon.

Dr. McCann said that, like most of the Fellows of the Society, he rather expected to hear a discourse on the general treatment of puerperal eclampsia, but the discussion had necessarily crystallized itself down to the two points brought forward by Dr. Herman—namely, the effect of emptying the uterus, and the effect of tepid baths. Probably the belief in the efficacy of the first depended much upon the theory that the pregnant uterus exerted pressure upon the renal vessels. But that theory was not now so strongly held, because where a much larger abdominal tumour existed, such as a very large fibroid, there was no pressure upon renal vessels nor consequent albuminuria to any great extent. It was

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interesting to find that the older obstetricians, such as Tyler, Smith, Sir James Simpson, and others, did not recommend the rapid emptying of the uterus. They advised general treatment and watching the condition of the patient, and if the fits became very severe, they recommended rapid delivery. All would agree that there was probably no more important sign, from a prognostic point of view, than very high temperature in cases of eclampsia. He did not think he had ever seen a patient with exceedingly high temperature in eclampsia recover. With regard to the case cited by Dr. Herman, he noticed that the patient had also received a saline injection, and was at the same time treated by morphia. It seemed to him difficult in that case to ascribe all the improvement to the bath.

With regard to the statistical table, of course it was very easy to prove one's case by figures, just as he believed it would be possible, by a series of statistics, to prove that rapid delivery did exercise an influence on the severity of the fits. It was well known that fits did continue after delivery, and indeed might occur for the first time after delivery. He would like to ask Dr. Herman whether he ever recommended that rapid delivery or the induction of premature labour should be undertaken in any given case—for instance, that of a patient who towards the end of pregnancy had albuminuria, diminution in the excretion of urea, scanty urine, and albuminuric retinitis, with headache, giddiness, vomiting, and œdema of the hands and face.

Dr. Gow said that in discussing the treatment of eclampsia it was important to remember (1) that we were entirely ignorant of the true nature of the disease ; and (2) that, apart from all treatment, a certain number of cases got well, whilst a certain number died. Does the treatment usually adopted lead to the recovery of cases that otherwise would have died, or to the death of those that otherwise would have recovered? In a disease such as eclampsia, which is so terrifying to the bystanders and unnerving to the physician, the patient is apt to be treated, if not with success, at least with energy. Treatment, when not founded on exact knowledge, is wont to be founded on hypothesis, and the working hypothesis in this case is that the disease is due to some poison or poisons circulating in the blood—in other words, that it is a toxæmia.

Theoretically speaking, the treatment should be directed either to eliminating the poison or to destroying or neutralising it by some antidote. No antidote is known, and the methods of elimination are uncertain. Failing these two plans, we may fall back on drugs such as morphia or chloroform, which, by lowering the excitability of the nerve-centres, render the fits less frequent, and appease the bystanders, if they do not benefit the patient.

The actual induction of labour must always do harm, although the completion of delivery may be advantageous. Dr. Gow agreed with Dr. Herman that the interference with the natural course of labour was to be avoided. Violent purgatives, such as croton oil, he considered objectionable. Over-activity in treatment should be guarded against, and until we know more of the nature of the disease it was better to adopt an attitude of rational watchfulness, and so avoid doing the patient harm instead of good. Some patients who had recovered might well feel grateful that they had, like the ancient mariner, escaped a twin danger—the Scylla of the disease, and the Charybdis of treatment.

Dr. Campbell Pope referred to the fact that the majority of cases of puerperal eclampsia which occurred in the practice of his father during

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a period of 60 years took place in one small village of less than 200 inhabitants. In that village there were three times as many cases of that disease as in the other 16 parishes in which he practised. He did not know whether that was still the case, but he suggested that it might be worth ascertaining from the medical men now practising there. Possibly it might throw some light on the causation of the disease. His father's method in such cases had been to get the labour over as quickly as possible, and he also employed bleeding, which seemed to afford temporary relief.

Dr. POLLOCK said Dr. Herman had distinctly brought out the point that, whatever was done, it was very undesirable to adopt the accouchement *forcé*. If one made up one's mind that it was undesirable to deliver, surely it was unwise to lacerate and tear the patient. But when a similar discussion was taking place some mouths ago before a kindred Society, one or two members urged that the accouchement *forcé* was the proper treatment under the circumstances. Delivery at all costs seemed to be what was advocated. He thought Dr. Herman had proved that such was not the case.

Mr. HUTCHINSON, jun., asked whether surgical treatment applied to the head of the woman, instead of to the uterus, had ever been tried. He had seen the marvellous effect of trephining in convulsions, and he had done it on the recommendation of a physician, but without believing it would have any good effect. But the patient got straight away well, and the temperature fell after the evacuation of cerebro-spinal fluid. Undoubtedly the worst cases of the disease were those in which the temperature rose; they all knew that the heat-centres in the brain were widely distributed, and that nothing was more likely to cause such temperature than increase of pressure on the cerebro-spinal fluid. The case upon which he operated was not one of eclampsia, but of unexplained convulsions in a male. Trephining on one side was a much less dangerous operation than many operations which were performed on the uterus; moreover, it could be done very quickly. In a case in which the temperature was going up in spite of baths, and in which the obstetric physician considered the patient must die, would Dr. Herman suggest trephining of the skull?

Dr. R. A. GIBBONS said those who had perused Dr. Herman's writings knew what an immense amount of attention he had given to the subject. The remarks he (Dr. Gibbons) would make would be directed to treatment, because the etiology was at present unknown. Dr. Routh had called attention to the fact that it depended upon some toxic agent, but whether that was due simply to defective metabolism, or to some peculiar secretion of some of the glands poured into the general circulation, was not yet settled. At any rate, the poison was strong enough to produce an acute and rapid degeneration of the cells of the large glands, such as the liver and the kidneys. Some time ago he read that the disease depended upon carbamic acid, which was secreted by the liver and poured into the general circulation. He had not since read anything supporting that. In some cases the dose of the poison found was so great that it rapidly killed the patient, while in others it was less, and there was a tendency for her to get well. In treatment, such drugs as chloroform, chloral, bromides, and morphia were of the greatest influence in calming the nervous system. But they did not cure the disease. He believed that anything which would dilute the poison would do good to the patient,

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and until something better was known he would continue to use the saline injection which he had employed, combined with bleeding. Though there were some cases in which, after the first rally from the injection, there was a relapse into coma and the termination was fatal, yet there were others in which the injection did great good.

With reference to the emptying of the uterus, he had felt more comfortable when the uterus was emptied. In the face of the statistics of Dr. Herman, it would be ridiculous to say there was any advantage in forcible delivery. He had noticed a convulsion come on with the onset of a pain, and therefore with the uterus emptied. When this could be safely and judiciously accomplished, one source of irritation was gone. To no disease could the adage "Prevention is better than cure" be more aptly applied than to puerperal eclampsia. He believed in many cases, if the urine were carefully examined systematically, there might be a great saving of life. He had no hesitation in saying that if a patient passed a large amount of albumin, even without other symptoms, the proper treatment was the induction of premature labour. He believed Dr. Herman and others would agree that where there was a large amount of albumin present it was impossible to predict the final outcome of the illness. He had seen cases of marked albuminuria without any other symptoms of disease. In one case of the kind in which the patient seemed well he recommended that procedure, but his advice was not accepted. In three days the patient was dead, having been apparently perfectly well up to within a few hours of the convulsion. His advice was, "Keep a careful watch on the urine."

Dr. Blacker said that Dr. Herman's paper was of the greatest interest, and particularly so to him, because it confirmed an opinion that he had long held. If eclampsia was due to the presence of some toxins in the blood of the mother, then the plan of emptying the uterus when the fits had already begun could not be of much value. How could the presence or absence of the fœtus in utero have any appreciable effect upon the amount of the toxin already circulating in the blood of the mother? He therefore thought that it was best to leave the uterus alone. The emptying of the uterus might, however, act beneficially by removing one of the reflex stimuli which set up the fits. If the toxic theory of the cause of eclampsia was accepted, then he thought that there were three main indications for treatment: Firstly, to lessen the reflex excitability of the nervous system by morphia, so as to assist the convulsions; secondly, to remove as much of the poison as possible from the patient's body without increasing the danger by venesection; and thirdly, by the transfusion of saline fluid, to so dilute the remainder of the poison as to lessen its injurious effects upon the tissues, and to promote its subsequent elimination by the kidneys and other organs. In a woman of average weight—say, 120 lbs.—the withdrawal of 500cc., or 17 ounces of blood, would amount to the removal of one-eighth or one-ninth of the total quantity of blood in the body, and a corresponding proportion of the poison circulating in the blood. By the subsequent transfusion of 1,000cc., or 30 ounces of saline fluid, the remainder of the blood in the body would have been diluted by one-fourth of its volume. The plan of treatment had been adopted in the last five cases under his care. Of these, four had recovered and one died. The treatment of eclampsia by vaginal or abdominal Cæsarean section seemed to him to be on a par with the recent proposal to treat cases of placenta prævia by a similar operation, a proposal which he held to be quite unjustifiable.

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Dr. HANDFIELD-JONES said that in the main he agreed with Dr. Herman, and he had never practised the method which Dührssen advocated. But the subject had been somewhat extended during discussion, and the question had been raised whether it was ever good practice to empty the uterus prematurely. In any discussion on puerperal eclampsia two cases always came vividly before his mind. The first of these was that of a young woman who married, and went to live with her husband in Ceylon. When between five and six months pregnant she developed puerperal eclampsia. That ended in natural abortion, and she recovered. Three years later she again became pregnant, and though she carried her child a little longer, eclampsia again developed, and was so severe that the husband said she should not become pregnant again. Six years later, however, when they were back in England, she became again pregnant, and at the sixth month was again seized with severe eclampsia. He emptied the uterus, and once more she struggled through. In the last year he had had another case under care, that of a fine, strong, healthy woman, who had been pregnant four times, and each time the pregnancy had terminated prematurely with eclampsia. On the last occasion the urine was watched week by week for the whole time, and it was noticed that any little error of diet or excessive fatigue produced marked albuminuria. It was only by the constant use of saline purges and other remedies, with careful dieting, that one was able to keep the urine in a normal state. As in the repeated pregnancies the albuminuria had always been noted as appearing in the third or fourth month of the pregnancy, and as becoming more marked as the gravid condition continued, until at last puerperal eclampsia appeared, one was driven to the conclusion that both those women had kidneys which, though capable of doing their ordinary work, were unable to bear the extra strain of pregnancy and the placental circulation. They were cases of renal inadequacy, such as Sir Andrew Clark brought forward. If that view were taken, then emptying the uterus and thus lessening the work of the kidneys would be common-sense practice. He believed there were cases in which the emptying of the uterus was the right practice, and although he did not believe in doing so by Cæsarean section or the method of Dührssen, yet the more rapidly and the sooner it could be done the sooner the woman would be in safety. He would controvert the theory that because the woman had fits after the emptying of the uterus, therefore that emptying was of no good to the patient. That was not sound reasoning, because, although there was sufficient poison left in the body to produce the fits, nevertheless the patient had been brought nearer the line of safety by removing the cause which was throwing the extra strain on the mother's kidneys.

He thought all would agree that one could not go over the notes of 100 cases of puerperal eclampsia without noticing an enormous difference in the clinical features of each case. There were cases in which the fits came on in the middle part of pregnancy and continued at distant intervals, but never assumed the virulent type. In such cases, by the injection of morphia and chloral, and by helping the kidney elimination, the patient could be got through to full time. But other cases were much more violent, and in those it might be that the emptying of the uterus was the only course to put the patient in a position of safety. In the main, he certainly agreed with the views advocated by Dr. Herman, but he would not like to leave the impression that he was in favour of abstaining from emptying the uterus in all cases.

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Dr. HERMAN, in reply, said the discussion had centred partly on his paper, and partly on collateral issues. Dr. Gow had rightly said we knew little about the causes of eclampsia, or what was the best thing to be done. But there were writers of text-books who said they did know a great deal, who knew that after delivery the fits at once ceased, and that by delivering the patient the fits were cut short. He was sorry none of the writers of the text-books he had referred to were present, as he would have liked them to have given some reasons for their statements, in the face of the figures which he had brought forward. One speaker described a case of renal disease during pregnancy, and inquired whether he (Dr. Herman) would induce labour in such a case. Undoubtedly. He regarded that as the right and proper treatment of renal disease in pregnancy which was not tending to recovery. He did not induce labour in every such case, because in many, by keeping the patient in bed and on milk, the symptoms disappeared. The nearer the patient was to term the less reluctant one should be to induce labour. The induction of labour as a prophylaxis against puerperal eclampsia was right practice. Perhaps he was wrong in postponing it as long as he did. But it was theoretically asserted that when the patient was delivered the fits at once ceased, and if so Cæsarean section, or vaginal Cæsarean section, would be the right thing to carry out. At one time bleeding was regarded as the right treatment. Ramsbotham said bleeding was the sheet anchor, and if one looked at reports of cases by the old writers one found that in nearly all of them the bleeding was followed by temporary improvement in the symptoms. The objection was that the bleeding so exhausted the patient that she died.

Dr. McCann asked whether it could be proved that the severity of the fits diminished after delivery. Nearly all the authors he had quoted said it did. The most common time for the fits to start was 12 hours before delivery, and in many cases the disease ran its course in 12 hours, the fits improving after delivery. The average mortality of eclampsia was about 20 per cent. Dührssen said that in 80 per cent. delivery either stopped the fits or improved them. That was a mortality of 20 per cent. in which he admitted his treatment did no good whatever—*i.e.*, his cases showed just the average mortality. According to him, the 80 per cent. improved because they were delivered.

He was much interested in Mr. Hutchinson's reference to trephining, and if he had a bad case in hospital he would see that Mr. Hutchinson was sent for. He was also interested in treating the hyperpyrexia by baths. There seemed to be few cases in the literature of the subject which were so treated for the high temperature, though the procedure was followed for producing diaphoresis.

With all respect to Dr. Handfield-Jones, he thought that if the fits recurred after delivery that showed that the delivery had not much effect on the fits. The induction of labour was a most excellent means of prophylaxis against eclampsia, but when the disease was developed, forcible delivery did harm rather than good. He thought the doctrine that one must interfere was a local expression of the general feeling that when a patient was very ill one must do something.

He hoped the discussion which had ensued would result in showing what a large opinion there was against the doctrine he had outlined, and that the authors he had referred to would be stimulated, by reading the discussion, into bringing forward some evidence in support of their statement that after delivery the fits at once ceased.

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April 28th, 1902.

THE QUIESCENT OR LATENT PERIOD IN THE COURSE OF GRAVE ABDOMINAL INFLAMMATION.

By A. H. Tubby, M.S. Lond., F.R.C.S. Eng.

In cases of acute abdominal lesions, where the diagnosis is in doubt, the simplest thing to do is to perform an abdominal section; but, as surgery is a science as well as an art, all are agreed that no exploratory operation should be performed without justification. There is, however, a debatable ground—a period of repose or quiescence—where instinct or intuition tells us that the patient is suffering from a grave abdominal lesion. Year by year, as these affections are more closely studied, and as clinical and pathological data accumulate, the debatable ground becomes gradually narrowed, but every surgeon from time to time in the course of his experience meets with cases in which he has conscientious doubts as to whether he should operate or should withhold his hand. Unfortunately, too, the effect of not sufficiently grasping the true state of affairs is more disastrous in this region than elsewhere, and cases can be recalled in which, after an acute onset, the pain subsides, the vomiting ceases, the temperature falls to normal, and the patient expresses himself as being tolerably comfortable. But subsequent experience shows that this change in the symptoms is by no means significant of real improvement. Sometimes this period is a deceitful and treacherous calm, and as likely as not marks the onset of grave complications which terminate fatally. It is this interval of apparent remission in the course of the disease which I venture to term the "quiescent or latent period"—quiescent in so far as the disappearance of some of the symptoms is concerned, and latent in that it precedes an aggravation of trouble, acute peritonitis, and a fatal issue.

I was much struck by a case which I was called to see some years ago. It was that of a married lady, aged 30 years, who, with a history of somewhat ill-defined dyspepsia for some years, was suddenly seized with acute pain about the umbilicus on a certain Monday afternoon. The pulse quickened, the temperature rose to 100·4° F., and the abdomen rapidly became tympanitic. On

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the next day the symptoms remained much the same until towards evening, when a marked improvement took place. I was asked, however, to see her in consultation on the Wednesday. I found her free from pain, the pulse and temperature normal, the facial expression placid, and apparently there was no need for any surgical interference. She was, however, kept in bed and carefully fed by rectal enemata, as it was suspected that leakage might have taken place from a gastric ulcer. On the Friday the symptoms returned suddenly and with great violence. I was called to see her in the evening and prepared to operate. She had been placed under the anæsthetic when suddenly she vomited and brought up a large quantity of blood, and life was soon extinct. Unfortunately no *post-mortem* examination could be obtained. The period of calm in this case, which occurred from the Tuesday night to the Friday morning, is an example of what I mean by the quiescent or latent period in acute abdominal affections. I do not myself doubt that there was some one symptom present, more or less marked, that escaped my attention, which, had I known it, would have revealed the full significance of the first onset; and if I had been called to the case at this time I should have examined the blood for leucocytosis. It is probable that, when the attack first came on, an ulcer had eaten its way down to the peritoneum, and a little leakage had occurred, the aperture being plugged by inflammatory lymph, but the ulcerative process went on rapidly until a large vessel was eaten into. And, looking backwards on the case, there is no doubt that the patient ought to have been operated upon during the quiescent period.

It must be confessed, however, that it is extremely difficult to be always wise at the time, and in contrast with this case I will allude to another gastric case. About three years ago I was asked by Dr. B. N. Dalton, of South Norwood, to see a case with a view to operation which he had diagnosed as perforated gastric ulcer, the onset having occurred about 18 hours previously. We operated, found the perforation, and applied an omental graft, and the case did quite well. About eight months subsequently, in Dr. Dalton's absence on his holiday, I was sent for again to see this patient. She had had rather a fatiguing day in London and, being hungry, had eaten some ham sandwiches rather late at night. In the early morning she was seized with sudden pain and some vomiting, and I saw her the same afternoon. When I arrived I found some

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tenderness over the upper part of the right rectus, and rigidity of the muscle there, but no tympanites, and the liver dulness was normal. On the occasion of the previous attack the liver dulness had been lost. I therefore decided to postpone the question of operation and to watch matters carefully, and, as events proved, that was the right course to be taken, for she has, so far as I know, never been troubled since with her stomach in any way.

But while the quiescent period was fairly well marked in the first of the stomach cases which I have quoted, yet there are many cases other than gastric in which it is better marked, and I should be disposed to say that the quiescent or latent period is from time to time a prominent feature in the following group of cases:—1. Injury to the intestines, with or without rupture. 2. Perforation of the intestines by ulceration 3. Injury to, and bruising of, the peritoneal connections of the viscera, with interference with their blood supply. 4. Acute diseases of the genital organs, especially of the Fallopian tubes. 5. Acute appendicitis, either ulcerative, perforating, or gangrenous, and this disease undoubtedly affords by far the greater number of examples of apparent improvement. In order to give point to my remarks, I should like to quote some cases from surgical literature:—

1. *Traumatic Rupture of the Intestine.*—Dr. J. J. Buchanan * reports the case of a young man, aged 18 years, who was struck on the abdomen at five o'clock one morning by an iron implement. When seen five hours afterwards he was unable to say where he had been struck. No ecchymosis, no sign of contusion, no area of tenderness was present. His general condition was good, and his pulse was of good volume, regular, and 88 to the minute. The temperature was 98° F. There was no abdominal distension nor excessive rigidity of the muscles, but the abdominal wall had not its normally soft condition. He had vomited after the accident, and once after admission to the hospital. Dr. Buchanan, on examining him, noted that peristalsis was almost absent, and on this symptom, taken with the slight rigidity and the fact that he had vomited twice, abdominal section was decided upon and was performed at 11 a.m. (six hours after the accident). On opening the peritoneum, thickened and congested bowel presented itself, and sero-purulent fluid escaped. On further examining the bowel

* 'Annals of Surgery,' vol. xxxii, p. 673.

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a rupture of the small intestine, about a third of an inch in length, was discovered with fæcal matter exuding. This was sewn up, the peritoneal cavity was douched with saline solution, a drainage-tube was put in, and the patient ultimately recovered. At the same time it must be admitted that there are many cases in which there is marked evidence of contusion of the abdominal wall with considerable collapse which recover entirely without operation.

2. *Perforating Ulcer of the Intestine.*—This condition affords excellent examples of this latent period. I quote a case described by Dr. A. B. Johnson,* in which a man, aged 27 years, was admitted to the Roosevelt Hospital with a history that four days before admission he was seized with colicky pains about the umbilicus, but there was no vomiting. His pains were not severe, and his bowels were moved daily, and he remained at his work. At 1.30 a.m. on the day of his admission he was seized with severe abdominal pains followed by a chill. On admission the abdomen was not distended. It was markedly tender, and was rigid throughout. Eight hours after admission the abdomen was opened and a good deal of cloudy, bile-stained fluid, with flakes of coagulated lymph, escaped. The vermiform appendix was examined, but was found to be healthy, and ultimately upon the anterior surface of the descending portion of the duodenum a small perforation was found forming the floor of an ulcer. The perforation was closed, and the patient recovered, and six weeks afterwards he was in the best of health. In this case the quiescent period, or, at any rate, the period in which grave symptoms appeared to be absent, lasted for four days.

3. *Injury to, or Bruising of, the Peritoneal Connections of the Viscera.*—Still more deceptive than the cases which I have quoted are those where there has been injury to the blood-supply of the intestine, or bruising of the peritoneal connections. As an example of this I may quote a case which occurred while I was a dresser. It was that of a middle-aged man who was knocked down in the street by an omnibus, the front wheel passing over him obliquely from the pubes to the left lumbar region. He was much collapsed when brought to the hospital, but there were no distinct symptoms. After three or four days he recovered, and it was thought that he might be discharged, but that experienced surgeon, the late Mr. Davies-Colley, preferred to watch the case, and on the eleventh

* 'Annals of Surgery,' vol. xxx, p. 634.

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day after the injury the patient was suddenly seized with acute symptoms of peritonitis, and he died in a few hours. On examination it was found that a considerable portion of the great omentum had sloughed.

A very remarkable case of traumatic rupture of the mesenteric arteries is reported in the 'Annals of Surgery' for March of this year. A very heavy man, aged 53 years, slipped and fell, and sustained a fracture of the bones of the leg, and some symptoms of shock were noticed. There was no pain in any other part of the body. On the following day the patient seemed weak, but was easy and cheerful. His temperature was 99·5° F., and the pulse was 99. The urine was scanty, but it contained no abnormal constituents. On the following day his condition had altered for the worse, and it continued to do so during the next four or five days, but no definite diagnosis could be made. There was some slight distension of the abdomen, but careful percussion revealed no localised dulness or undue resonance. He was extremely pallid, and breathed irregularly; his temperature was sub-normal, and his pulse was 160, and it was thought that he was dying from internal hæmorrhage. On the seventh day after the accident he died, and on opening the abdomen a large quantity of blood was found, which was due to the ruptured walls of several of the mesenteric arteries. The mesentery contained an enormous amount of fat, and there can be no doubt that in the pull exerted upon this structure by the violent fall, it and its vessels were lacerated. In this case the latent period may have been due to the slow hæmorrhage and absorption of the liquid portions of blood in the early stages of the case by the peritoneum.

4. *Acute Diseases of the Genital Organs.*—The records of gynæcology furnish numerous examples of acute diseases of the genital organs, especially of ruptured Fallopian tubes from pyosalpinx and tubal pregnancy, where the serious nature of the disease has not been at once evident.

5. *Acute Appendicitis.*—The latent period, or, as Mr. Charters Symonds,* in his very able and instructive Hunterian Oration designated it, "the period of repose," is extremely well marked in cases of appendicitis. He mentions one case of a female, aged 20 years, who was seen 48 hours after an acute onset, and was considered so well on arriving at the hospital that operation was

* 'British Medical Journal,' 1899, vol. i, p. 520.

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postponed until the next morning, when a foul abscess was evacuated, and he says that "in nearly all the acute cases a stage is reached soon after the onset marked by a cessation of all acute symptoms. The patient passes, unaided by sedatives, into a condition of repose. This period, if wrongly interpreted, may lead to disaster. It is in its early stage a period of safety, one in which operation is successful." I can give two cases from my own experience. One was that of a young woman, aged 18 years, who after a day in town, where she had partaken of a couple of ices and later in the day of a raw apple, was seized early on the following morning with acute pain and vomiting and sickness. This subsided after 24 hours, and her condition improved during the next 48 hours, so that she was able to get up and to go about almost as usual. On the fourth day intense pain in the abdomen set in, and vomiting returned, and she was prostrate. The parents were urged by the medical attendant to have surgical assistance, but this was refused until the fifth day of the illness, and when I saw her the condition was that of acute septic poisoning. The temperature was 100° F., and the pulse was 124. The abdomen was distended, with no sign of tumour or dulness, and, in fact, she presented a picture of virulent peritonitis. As a remote hope an operation was undertaken, and a long, much distended appendix was found, but her condition became so bad that it was not possible to remove it. The abdomen was washed out with saline solution, and the appendix was packed round with strips of iodoform gauze. For 12 hours she showed a marked improvement, but then aggravated symptoms set in and she succumbed 24 hours after the operation. The other case was that of a young man, aged 18 years, who, after a bicycle ride on a certain Sunday, was seized with acute abdominal pain while away from home. After 24 hours, however, he felt well enough to return home, and he appeared to be doing well, and remained in fair condition until 2 p.m. on the following Tuesday, when the pain and vomiting returned, and his general condition was that of collapse. I saw him at 4 a.m. on the Wednesday and found his pulse uncountable, his temperature 101°, his abdomen distended, but with tenderness and an ill-defined mass in the right iliac fossa. On opening the abdomen I evacuated some foul pus from the right iliac fossa. After the operation his pulse improved somewhat, and he expressed himself as being free from pain, but he did not survive the operation for more than 24 hours.

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These are instructive instances of the subtlety of these cases, but the most striking cases that I have ever read of are those quoted by Sir Frederick Treves in his work on 'Perityphlitis,' at p. 27. He says that he has seen two cases of septic peritonitis, due to trouble in the appendix, run their entire course with a flaccid abdomen and with scarcely any tenderness. Both patients were men over 50 years of age. No morphia had been administered, and in both cases death took place from toxæmia within seven days. One man spent some hours in applying massage to his own abdomen, and the other drew frequent attention to the absence of tenderness by slapping the abdominal wall. In all these instances, although the usual train of symptoms of virulent mischief was not present, yet there was some one sign or other which subsequent investigation showed to afford sufficient grounds for surgical interference without delay. It may have been either that the remissions were never quite complete, or that the usual course of things differed by the persistence of some symptom. Thus muscular rigidity—slight it may have been—may have persisted, peristalsis may have been absent, some tenderness may have been elicited on careful palpation, the pulse may have been out of proportion to the temperature, or the general appearance of the patient—deceptive though it may be to the casual observer—may have suggested to the experienced eye that all was not well. Again, it is extremely difficult to gauge the value of symptoms or signs slightly marked when the deceptive remission has been brought about either by drugs or by the application of ice-bags. As to what is the explanation of this quiescent period it is difficult to say, but in some cases where tenderness has been absent—as, for example, in cases of very acute appendicitis—it has been found that the appendix is gangrenous, and that its nerves are, therefore, insensitive. And so it may be that in cases of acute general peritonitis the sensory nerves of the peritoneum are paralysed from the first. Doubtless there are other causes at work, but it is not within the scope of this paper to discuss them.

A careful compilation of abdominal cases, and the recording of clinical signs, fortunately enable us to fix upon certain symptoms—even in obscure cases—as of great moment:—

1. The most important information is gathered from repeated records of the *pulse and temperature*. These may rise together in due proportion; or there may be a slightly elevated temperature

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with an entirely disproportionate pulse-rate; or the temperature may be subnormal when taken on the surface, while the pulse may be greatly accelerated. It is this last condition that is more often met with during the latent period in very grave cases, and taken with a history of the onset of the attack it affords justifiable grounds for immediate operation. In all abdominal cases the pulse and temperature should be frequently taken, at least every three hours, although the disease appears to be quieting down. Remembering what has been said as to the probability of a successful issue to an operation, if it be undertaken in the early part of the latent period, this inverse relationship of temperature and pulse should be observed at the earliest possible time. If the altered relationship has been established for a day or two, then the prospects of operation are extremely unfavourable, because by that time the patient has probably become saturated with septic products. Again, it may happen—as, for example, in the course of an attack of acute appendicitis—that after an initial rise of temperature and pulse there is a fall in both, succeeded after a few days by a rise. This indicates the presence of a considerable amount of pus, and unless the abscess be evacuated there is a grave danger of it bursting into the peritoneal cavity.

2. In the case of the perforating duodenal ulcer which I have quoted, there was no vomiting nor severe pain, and the patient was able to remain at his work, *rigidity*, with some tenderness, being the only definite symptom; but, on opening the abdomen, it was found that there was acute peritonitis present, and, perhaps, of all the physical signs, a transient or a persistent rigidity, and the absence of other symptoms, are the most valuable, and they become convincing when signs of intestinal peristalsis are absent.

3. It is difficult to speak of *distension* as a symptom taken by itself, for it is often absent in the early stages, even in so serious an affection as a perforating gastric ulcer, but in the late stages of abdominal diseases the fatal significance of it is only too well known. But distension which persists for longer than for from 24 to 36 hours, and is unrelieved by treatment such as a turpentine enema, is of grave moment.

4. As a rule, *vomiting* is quite absent during the quiescent period. It is true that at the beginning of the illness there may have been vomiting, but at the "period of repose" the vomiting has subsided; it may be because the stomach has been emptied of its contents by

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the initial sickness, or because the vomiting has been kept in check by the administration of sedatives. But if the vomiting recurs in spite of careful feeding, careful nursing, and more especially if it persists after the administration of sedatives, the symptom is not one to be under-estimated.

5. A great deal of careful work has been done on the subject of *leucocytosis* of late, and not only on leucocytosis, but on the condition of the blood generally in abdominal affections. Dr. Da Costa* states as the result of his researches, that in an appendicular abscess the number of leucocytes per cubic millimetre present in the blood may be from 15,000 to 20,000, the normal number of leucocytes being from 5,000 to 7,000 per cubic millimetre, and rising to 12,000 at the height of digestion after a good meal. He further adds that if the pus focus be walled off, and no absorption of toxic material takes place, then there may be no rise in the number of leucocytes, but otherwise in purulent appendicitis their number may vary from 14,000 to 58,500, and he found the average in purulent cases to be 17,450. In perforative appendicitis there is a very abrupt rise of leucocytes, and counts of over 20,000 indicate either pus, gangrene, or peritonitis. If the number be under that the deduction is uncertain, for in 35 per cent. of non-purulent cases there is merely a small increase in leucocytosis. As showing, however, that even this sign is not to be absolutely relied upon, Dr. Da Costa states that in some septic cases arising from appendicular trouble there is an absence of leucocytosis, and if this occurs it is a symptom of very grave importance. From a diagnostic point of view it should be remembered that leucocytosis is absent in typhoid fever, but is almost invariably present in ovarian abscess, pyosalpinx, perinephritic and nephritic abscesses, and in suppuration generally. Dr. J. T. Finney,† writing on perforating ulcer of the stomach, quotes Dr. Cabot, who states that leucocytosis is never seen in the course of gastric ulcer, except after severe hæmorrhage and at the height of digestion. The presence of leucocytosis, when the influence of bleeding and digestion is excluded, is "practically unknown in uncomplicated gastric ulcer." When, therefore, grave symptoms occur in a young woman with a history of previous digestive disturbance, together with a sudden development of marked leucocytosis, the indications for immediate surgical interference

* 'American Journal of the Medical Sciences,' vol. cxxii, p. 645.

† 'Annals of Surgery,' vol. xxxii, p. 6.

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are imperative. A most instructive article on the leucocyte count in surgery is given by Dr. Theodore Dunham.* He gives many interesting details, some of which are as follows:—There is considerable leucocytosis after the administration of ether. In acute cervical adenitis in an infant, aged 7½ months, the leucocyte count was 9,800, and with a mastoid abscess 24,800, and with a gangrenous appendix 17,000, rising next day to 19,000. In another case the appendix was operated upon, and the day after operation the count was 11,700, and on the following days it was 14,500, 16,900, and 22,100. Meanwhile the dressings, the temperature, the pulse, and the respirations gave no distinct indication of anything wrong, but at the dressing after the last-named count a large quantity of pus had found its way into the wound.

Mr. J. H. Hebb has very kindly made some observations for me at the Westminster Hospital upon leucocyte counts in various conditions, suppurative and non-suppurative. A male patient was admitted suffering from appendicitis, with a temperature of 99° F. The leucocyte count was 11,562 (finely granular 54, coarsely granular 17, hyaline 9, and lymphocytes 20 per cent.). This case subsided without suppuration, and did well. A female patient was admitted on February 15th suffering from suppurative pelvic cellulitis. The leucocyte count was 22,500. On March 1st, when the suppuration was subsiding, the count was 15,625. A female patient suffered from pelvic cellulitis and a very extensive abscess on the outer side of the thigh, which had made its way from the pelvis through the great sciatic notch. The temperature was 98° F. On admission leucocytosis was 13,128 (finely granular 43, coarsely granular 4, hyaline 10, and lymphocytes 43 per cent.). On March 3rd, when the suppuration was less, the count was 11,560 (finely granular 64, coarsely granular 5, hyaline 6, and lymphocytes 25 per cent.). So far, then, it may be said that in the leucocyte count frequently made in the course of the illness there is a very valuable indication that grave mischief is going on, even when the other symptoms were quiescent or latent, and this method of clinical investigation promises to be one of the utmost value.

6. In regard to *chest complications*, in a patient who began his illness with symptoms referable to the abdomen, but who remains ill, although the symptoms in that region have apparently subsided, careful examination of the chest and the detection of either a

* 'Annals of Surgery,' vol. xxxi, p. 669.

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pleuritic rub or the presence of fluid in either of the pleural cavities may serve as an indication that mischief is still going on in the abdominal cavity, and that exploratory operation is called for. In Dr. J. Mitchell Bruce's excellent Hunterian lecture, delivered in November of last year, the subject is fully dealt with, and he quotes an instructive case of pleurisy and perigastric abscess, which was operated upon by Mr. Rickman J. Godlee, who found serous fluid in the left chest and a perigastric abscess below the diaphragm. A somewhat similar condition is met with in cases of appendicular trouble, where the abscess has tracked up into the renal regions, and associated with it fluid has been found in the chest. It may be that the trouble has almost, if not quite, subsided in the region of the appendix, and what has to be dealt with is a continuous lymphatic infection in the post-abdominal connective tissue, extending through the lymphatics to the diaphragm, and so causing chest symptoms. The detection of fluid in the chest, especially on the right side, may lead on exploratory operation to the further detection of a collection of pus, either sub-diaphragmatic or situated deeply in the flank.

The remarks which I have made on this difficult clinical subject are, I fear, fragmentary and imperfect, but if they lead to a more thorough investigation of that all-important period—latent, quiescent, or reposeful—I feel that what has been written may be of some service.

Dr. T. D. Savill congratulated Mr. Tubby on his excellent contribution, which was of very profound medical and surgical interest. All must have met with those very difficult cases in which there was something obscure in the abdomen, and if there were any means of bringing to light what was going on, he thought the utmost use should be made of them. During the last year or two he had met with a relatively large number of abdominal cases, in which both surgeons and physicians seemed equally perplexed; but if there was one clinical sign more than another which gave a clue as to the nature of the case, and the course it was likely to pursue, he thought it was the pulse-temperature ratio. If the pulse rate was over 100, even though the temperature might be subnormal, there was sure to be some mischief going on which needed prompt surgical aid. With regard to the leucocytes, he did not attach so much importance to the number of them as to their character. In an examination of blood films there was an almost certain sign of suppuration within the body. It was not so much the number of leucocytes as the number of polyneuclear cells. The average proportion of polyneuclear cells to the total white cells was about 65 per cent., but there never was suppuration going on in the body without there being an increase in the polyneuclear cells. That could be ascertained by an easier method than even the counting of the leucocytes. All that was

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needed was specially prepared cover-glasses with which to make the films, and at the present time there was a double stain, Leischman's stain, obtainable in England, which could be easily applied. It only required a few minutes to stain the films and count the relative number of polyneuclear cells. He had seen cases in which the proportion had gone up to 85 and 90 per cent. Anything above 70 or 75 per cent. invariably indicated a pus formation of some kind, which would probably require to be dealt with promptly and surgically.

Dr. Bryant said he was glad to hear Mr. Tubby draw attention to the great importance of blood examinations in obscure abdominal cases. It was a method of examination in which English practitioners were very backward. He thought that at the Johns Hopkins Hospital, in America, every case admitted was submitted to a blood count, quite irrespective of the diagnosis. In appendicitis, in particular, very great importance was attached to the result of that examination, and in many such cases in which there was a doubt as to operation, the blood count often decided whether it should be undertaken or not. In every case in the wards which he was looking after in Guy's Hospital he was having a blood examination made, and the results had been not only most interesting and instructive, but of the greatest help in indicating the advisability of operation. Some time ago he had two cases of appendicitis. Both were extremely ill, both had abdominal tumours, and the question of the advisability of operation arose. In one of the cases the leucocyte count was 11,000, and in the other case 27,000. The last case was operated upon, an abscess was found and evacuated, and the patient got well. In the other case the inflammation subsided without operation, and the patient came in subsequently and had the appendix removed.. At present he had a case of appendicitis in which the leucocytes were 10,000, and the patient was getting better without operation. 20,000 leucocytes or more certainly indicated the need of operation. With regard to pulse, his experince was that the pulse was very rarely low in abdominal cases, and he agreed with Dr. Savill that when the pulse rate was over 100 it was very important to operate. The pulse-temperature ratio was particularly important in cases where it was difficult to diagnose between appendicitis and typhoid fever. It was very rare to find leucocytosis in cases of typhoid fever. At present he had a case of this disease in which the leucocytes were 2,500 to the cubic millimetre. With regard to thoracic complications, perhaps it would be too sweeping to say that if pleurisy were present it would indicate immediate surgical interference, because great difficulty sometimes arose when dealing with diaphragmatic pleurisy which was not associated with any abdominal lesion. Recently he had a case of that nature, which he asked his surgical colleague, Mr. Dunn, to see, and it was decided to leave it without operation. A few days afterwards the patient developed pleurisy, from which he subsequently got well, without there having been any indication of the presence of serious abdominal disease.

Mr. Hutchinson, jun., said the quiescent period mentioned by Mr. Tubby referred to a condition which existed in two different kinds of cases. One was where there was a prolonged period of absence of grave symptoms, and the other in which grave symptoms came on, and then a quiescent period supervened. He thought most of the second class of cases could be explained by the influence of drugs or some poison on the

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central nervous system. But there were certainly great differences in the degree of reaction of the peritoneum. If that were not so, why should the symptoms in rupture of the intestine or stomach differ so extremely in different cases? He remembered a case in which an Italian organ grinder was being plagued by boys in the street, when the Italian pulled out his knife and stabbed one of the boys in the groin. A narrow wound passed obliquely upwards above Poupart's ligament, but there was a complete absence of all symptoms in the first few hours. But, owing to the knife being long and the Italian being strong, he thought it better to open the abdomen. He then found the small intestine had been wounded, and there was an escape of fæces to a considerable extent. He sutured the wound after excising its edges, and cleansed and drained the peritoneum. Undoubtedly the boy would have died but for this interference in the quiescent stage. Mr. Tubby had mentioned that some cases were to be explained by the nerves of the intestine being so damaged that they ceased to transmit any message, and consequently there was no reflex symptom. In one case of gangrenous gut in a strangulated hernia the patient insisted on walking to the operating theatre, and did walk some of the distance. At the operation the intestine was found to be gangrenous. There was scarcely any peritonitis. Probably the nerves had been practically cut across by the strangulation. He was glad Mr. Tubby pointed out that rigidity of abdominal muscles was not the infallible test of peritoneal inflammation that some writers affirmed it to be. In one case a lad had his duodenum ruptured at football, but the injury was followed by remarkably slight symptoms. The lad lived about a fortnight, and at one time it was thought he would recover. The intestine was extensively ruptured. As to leucocytosis, he asked whether Mr. Tubby's experiments had been made in many cases. He had seen it stated that leucocytosis was of little value in diagnosing the presence of pus in the abdomen. Possibly by extremely recondite researches it was possible to know, but it seemed hopeless to get such researches done quickly enough to decide as to operation by the ordinary working operator. Moreover, he was very sceptical as to the value of the leucocyte count in a doubtful case of peritoneal suppuration, as he had known it give a wholly fallacious result.

Dr. de Havilland Hall thought there had been too much stress laid on the connection between abdominal affections and pleurisy. They had all seen cases (especially in children) in which the first symptom of pleurisy was located in the abdomen. He had seen it in a considerable number of cases in which the medical man had suggested that the child had been suffering from peritonitis. On further examination, however, fluid was found in the pleural cavity. In the majority of those cases the pleurisy, though it might have been started in the diaphragm, certainly was not associated with any abdominal condition. Therefore, though in some cases of appendicitis and sub-diaphragmatic trouble there might be pleurisy with effusion into the pleural cavity, on the other hand there were cases of diaphragmatic pleurisy in which the symptoms at the commencement were distinctly of a peritonitic nature.

The President joined in the expression of thanks to Mr. Tubby for having formulated a series of points to which one's attention should be directed in that very anxious period when the physician and the surgeon had to decide as to the propriety of operation, whether it were in those cases which commenced with severe symptoms followed by a lull before

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bursting out again, or those in which severe symptoms were in abeyance from the first. It was very important to obtain some information of a reliable and easily ascertained character which should guide as to the propriety of recommending an operation; something, perhaps, other than the investigation of the leucocytosis, which required, at all events, some time, some apparatus, some experience, and some skill in manipulation. Of all the symptoms which had been referred to—pulse and respiration, rate-temperature, pain, muscular rigidity, &c.—the pulse-rate seemed to be the most important. It was not by any means the first time that that symptom had been drawn attention to in that room, and they were indebted to Dr. Caley for a paper read before the Society a few years ago in reference to the question of appendicitis, in which the importance of the pulse-rate in forming an opinion as to operation was insisted upon, more especially in cases where the symptoms were advanced. It was satisfactory to note that from a somewhat different point of view the importance of the same feature should be recognised.

Mr. Tubby, in reply, thanked the Fellows for the attention bestowed upon his paper. He could fully confirm the remarks of Dr. Bryant as to the Johns Hopkins Hospital. The extraordinarily good work done there would favourably compare with that done anywhere. He had no idea of conveying the impression that the pulse was slow in the conditions under discussion; the pulse was rapid and the temperature low. With regard to pleurisy, he thought that was in many cases by no means a simple uncomplicated disease. No one in a case of long illness which began in obscure abdominal trouble would be content with merely finding pleuritic fluid in the chest. It would naturally lead one to suspect that there might have been something arising from the abdomen. That mere suspicion had led to several brilliant results in the way of operative interference. He thanked Mr Hutchinson for drawing attention to two kinds of latent period. That touched upon a question which he would have liked to have dealt with, but it was sufficient to justify a separate paper—namely, the peculiar behaviour of the peritoneum in different regions. The explanation he wished to advance as to why in some cases there was a strong reaction to irritation of the peritoneum in one region and not in another was, that it was due to the distribution of the lymphatics. One broad fact stood out clearly—namely, that the nearer one approached the diaphragm the greater was the number of lymphatics in the peritoneum. In the cavity of the pelvis it was tolerant to all sorts of interference and suppurations, but higher up there was a great deal more lymphatic tissue, and the irritation spread from the peritoneum through the lymphatics of the diaphragm and set up pleurisy. He agreed with Dr. de Havilland Hall in the remarks which that gentleman had made.

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BILHARZIA HÆMATOBIA.

By Lieutenant LELEAN, F.R.C.S., R.A.M.C.

MR. PRESIDENT AND GENTLEMEN,—The subject which I propose to bring to your notice to-night is that of the Bilharzia Hæmatobia parasite. Until quite recently it was relegated by most English practitioners to the list of tropical diseases, robbed of practical interest by remoteness. My apology for obtruding it to-night is that this is no longer so. There is a constant—if small—stream of cases returning from South Africa and resuming civil life whose hæmaturia will call imperatively for diagnosis by the practitioner who is called in. Hence its recognition becomes a matter of moment, while its therapeutics afford a field attractive alike from unexplored possibilities and remarkable difficulties.

I propose to consider it :—

(1) Morphologically.
(2) Developmentally.
(3) Pathologically.
(4) Clinically.
(5) Therapeutically.

(1) MORPHOLOGICALLY.

The direct line of descent from sub-kingdom to family is through the Metazoa Platyhelminthæ, Trematoda, Malacocotylea, and Distomidæ.

The phylum Platyhelminthæ embraces the flat worms or flukes with a typical flat, oval, cilia-covered body, and the systems, shown in Plate I, alimentary, reproductive, excretory, and nervous. Of its three classes, the turbellaria live a free-swimming, vigorous, independent life; while the trematodes and cestodes lapse into parasitic habits. The first stage, that of ectoparasitism, is induced by feeding on mucus excreted by other species; in order to hold on more easily suckers are developed, and effort and speed being no longer required, the cilia are replaced by a chitinoid substance, secreted to protect the organism from the juices and juxtapositions of its host. The rudimentary eyes forfeit their existence by disuse, and eventually the cestodes, lapsing still further into endopara-

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sitism, lose all trace of alimentary tract, and become mere saccules of parasitic inertia—motionless, sightless, and organless.

The Trematoda are the suctorial worms, and are most commonly parasitic invertebrates of aquatic habits, a fact bearing on their development. Briefly, they are parasitic Platyhelminthæ, which retain the mouth and alimentary sac of the ancestral type—lose the cilia and gain suckers and a cuticle. Their classification by the number of suckers affords some interest:—(1) The Heterocotylea.

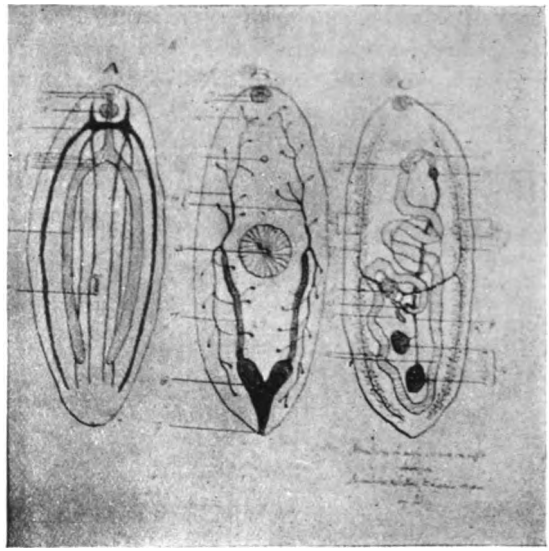

PLATE I.

A.—Nervous System (black) and Alimentary System (shaded) of Platyhelminthæ (schematic).

B.—Excretory System: (6) Flame cells; (8) Bladder; (9) Posterior excretory pore.

C.—Generative System: (2) Seminal glands; (3) Sperm duct; (4) Seminal vesicle; (5) Penis; (6) Vitellarium; (7) Vitelline duct; (8) Ovary; (9) Laurer's canal.

This order has one large terminal and two oral suckers. Being ectoparasitic, it needs more support to avoid being carried away during its host's movements; (2) the Aspidocotylea has one large ventral; and (3) the Malacocotylea, with which we are most concerned, has two suckers only, though they may be aided by the

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papillæ and hooklets as accessory adhesive organs. Of the Distomes little need be said, save that they are widely distributed in Africa, India, and the Far East as human parasites, but are rare in Europe.

Thus, reaching Bilharzia itself, I may first notice, in passing, two allied organisms—Bilharzia Bovis (of Sonsino) and Bilharzia Magna (Cobbold)—in the cava of cercopithecus fuliginosus. The adult worm is most difficult to obtain. It occurs, so far as is known, only in man and one of the cemidæ (an ape described by Sonsino), and not causing death, save in most rare cases, it remains inaccessibly in the radicles of the portal vein, beyond the reach of the pathologist. The male worm is usually a half-inch long, milk-white in colour, and presents a tuberculated cuticle. The lateral margins of the flattened body fold over, to form the "gynæcophoric groove," in which is enclosed part of the body of the female. This groove

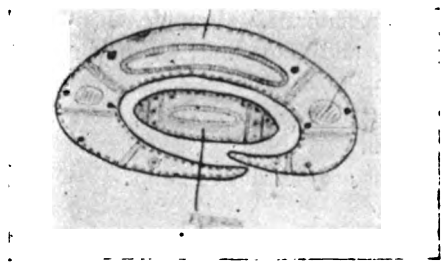

PLATE II.

Transverse section through the posterior portion of the male worm (A), showing the overlapping lateral margins forming the gynæcophoric canal in which the female (B) is seen lying.

extends from the ventral sucker to the tail, and is often converted into a canal by the overlapping of the margins, as is seen in Plate II. Near the anterior end is placed the mouth, with its peri-oral sucker; and a little posterior is the commencement of the gynæcophoric groove, lying just behind the ventral sucker, with the genital pore in its depths. The suckers are about $\frac{1}{100}$th of an inch across, and fix the head to allow the mouth free play. There being little strain in the portal vein, they are not provided with hooklets or spines; nor is it necessary to have any chitinous framework, as in the ectoparasitic Heterocotylea. The structure of the suckers consists of muscle arranged in longitudinal, vertical, and retractor trabeculæ covered by epidermis, they are never multilocular. The

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female is longer and finer than the male, resembling a silk thread, being about $\frac{4}{5}$ths of an inch long by $\frac{1}{90}$th at the broadest part, thinning away to $\frac{1}{300}$th at the filiform anterior extremity. She is quite smooth, and lies partly in the gynæcophoric groove, the genital pores being closely approximated. The male being tuberculated as a means of proceeding against the blood current, it is obviously unnecessary that the female should be similarly equipped. The female is invariably found paired when not encysted, and in many cases none can be found save those so encysted. The condition seems to be one of perpetual coition, save when the female is ovulating—the male holding on by his suckers, and the female being retained *in situ* by the enfolding of the lateral margins of the gynæcophoric canal of her consort. In both sexes the skin has lost the cilia of the ancestral type—becoming horny—the superficial epidermal cells losing their nuclei and resting on a thickened basement membrane. Beneath this is muscular tissue, longitudinal, circular, and trabecular, as shown in Plate V. The main bulk is made up of a mass of branching cellular mesoblast, in which the organs are simply embedded, there being no general body cavity or coelom.

The various systems are shown in Plate I.

1. *Nervous* (Fig. A).—Seen centrally, with a rudimentary bilobed brain in the prostomium, arching over the ventrally-placed œsophagus. It forms the sole ganglion, and is connected by five longitudinal nerve tracts with a general plexus by means of annular branches. Twigs from the plexus reach widely-distributed sensory cells and the individual muscle fibres. The system is derived by epidermal differentiation.

2. *Alimentary* (Fig. A).—The mouth, which serves also as anus, is placed at the anterior end of the ventral surface, surrounded by the peri-oral sucker. It leads through a well-developed globular sucking pharynx and a short œsophagus into a median enteron, which almost immediately bifurcates into two lateral forks reuniting posteriorly, to extend spirally towards a blind ending in the caudal extremity. In the fresh specimen the intestine is stained red by the contained blood. It will be seen that this is a deviation from the simple saccular type of intestine (shown in Plate I) of the ancestral Platyhelminth—a deviation the result of the simultaneous development of the genital organs and the flattening of the body,

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causing a dorso-ventral adhesion of the walls of the sac. That flattening is a factor is shown by the fact that in the Aspidocotyleæ, which show much less flattening, there is still a central saccular intestine. The requisite absorptive surface is maintained in the ectoparasitic Heterocotylea by the development of lateral cæca, but in Bilharzia the food is so absorbable that such increase of area is not necessary, and cæca are absent.

3. *Excretory* (Fig. B).—The excretory apparatus consists of terminal "flame-cells," (6) whence efferent twigs unite to form a pair of laterally-placed ducts (7), which join in a median contractile bladder (8) with a terminal posterior excretory pore (9). The "flame-cells" are so named because of the curious optical effect produced by the synchronous movements of the cilia in their hollowed interior. Developmentally they are derived from unicellular epidermal glands, whose stalks fuse, get detached from the surface and form longitudinal ducts by continued fusion and canalisation, the cells themselves meanwhile becoming hollowed.

4. *Generative.*—The common hermaphrodite form is shown in Fig. C, which is mainly applicable. The male Bilharzia has five seminal glands (2) leading by a sperm duct (3) through a seminal vesicle (4) to the penis (5), which lies in the antrum masculinum opening at the genital pore on ventral surface. The penis is eversible, the hooklets of the terminal part of the duct thus being turned out so as to afford a prolonged hold, the organ being almost certainly intromissary.

The female possesses two vitelline ducts (7), extending from the vitellarium (6) to join the oviduct just as it leaves the ovary (8). Near the point of junction "Laurer's canal" (9) extends from the oviduct to the surface opening on the dorsum. The ova pass the ootype where they are invested with a chitinous covering secreted by the surrounding shell-glands. Becoming the uterus at this point, the canal proceeds by a convoluted course to the antrum femininum, the contained ova lying in great numbers in the lumen, their spines directed backward. Morphologically, "Laurer's canal" is most interesting. It is not the analogue of the vagina, as has been suggested, for spermatozoa have been observed directed from the oviduct to the surface by ciliary action. It thus appears that it is an aspiration escape, the cilia drawing the spermatozoa up through the uterus to impregnate the ova before they receive their impervious chitinous covering in the ootype. The ovaries are derived from

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the wall of the archenteron by a process of tubulation. These structures and their relations are well seen in Plate II in transverse section, the two sections being in their normal relation.

Ova.—The ova of Bilharzia (Plate III) are in shape like elongated hen's eggs, averaging some $\frac{1}{300}$th inch, or 120 μ in length, the extremes being 60 to 150. They present at one end a sharp spine, and are frequently pigmented. The spines are not invariably terminal, some being laterally placed, and it was held at one time that these lateral spines were peculiar to ova from the rectum, those from the bladder being invariably terminal; but in a rectal case which has

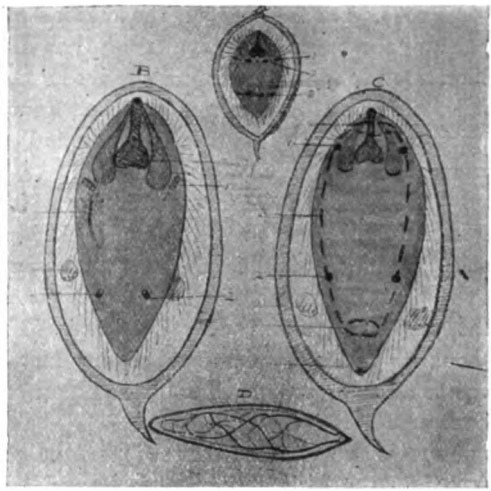

PLATE III.

Ova of Bilharzia: A and B showing various stages of development of the embryo, terminal spine, and chitinous shell; in C the cephalic and caudal vascular circles are seen in longitudinal and in D in transverse section.

been under my care they were all terminal, and I have occasionally observed vesical ova with spines on one side, a fact which has not been before noticed, so that there is apparently no essential difference dependent upon locality. The spines are homologous with the egg-appendage, which may, as in the Octobothrium Lanceolatum, occur at both ends, affording fixation points; analogous lateral spines are found in the ova of the Dactylogyrus. In the Bilharzia they serve the double purpose of preventing backward movement in the passage down the oviduct, and of affording fixation to the shell during the

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efforts of the embryo to free itself. The ovum consists of a transparent chitinous shell, 15 to 20 μ thick, resisting digestive agents, but soluble in 30 per cent. KHO. Immediately inside the shell is a delicate granular lining membrane enclosing a clear fluid, in which lies the embryo, usually with its head towards the spine end, while between the embryo and the membrane are a number of bright granules. Immature ova are only half the size of those that are mature: they are narrower, and the contained embryo consists of a structureless mass of granules. As development proceeds the head and cilia appear together, and the granules coalesce. The mature embryo is covered by a transparent dermis, beaded on the inner surface, and covered, save at the oral papilla, by cilia. The cilia are directed forwards in the anterior third, backward in the posterior two-thirds, and reach a maximum length of $\frac{1}{2000}$th inch at the shoulder, where the arrangement is vertical. They are arranged in regular transverse and vertical lines, and can be seen whirling the granules in ceaseless eddies. The head has at its apex the oral papilla, 10 μ across and cilia-free, from which radial ridges reach outward to the broadest part or shoulder, at the junction of the anterior and middle thirds, where a coronal line of apertures can be seen. Further back a second line of similar lateral apertures divides the posterior from the middle thirds. From these apertures Brock has seen in process of extrusion the bright granules that lie between the embryo and the shell; this I have never been able to confirm, and certainly many such granules are much too large to have been thus derived. Alternating with the anterior apertures are the fine ligaments, which attach the shoulder of the embryo to the lining membrane, and when torn they can be seen stationary amid the rows of waving cilia. Within the embryo can be made out the structures shown in Plate III. A rugous mouth leads to a stomach well-defined in front, but obscured behind, where its outline is lost under the mass of granules. It is described as bifurcating to subdivide and communicate with the posterior lateral apertures, but I am inclined to think it does not extend so far, as I have seen a clearly-defined limit posteriorly in one case when the movements of the embryo had momentarily displaced the overlying granules.

Another theory is that it communicates with the coccygeal dimple described, but if there be a coccygeal dimple it is most inconstant, as I have not yet found it present. On either side of the stomach, and on a level dorsal to it, is a clearly-defined rounded

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body—the "head-gland" of Coe, the "lemniscus" of Sonsino, the "pyriform body" of Cobbold—which is connected by a stalk with an elevated papilla beside the mouth. The stalk has, I believe, never been actually seen to be canalised, and the "nucleus" may be possibly the contractile body described by Brock in his able articles, in which he advances a theory that it is a muscular organ concerned in moving the head, on the observation that the structure is a granular matrix containing circular and longitudinal muscle, the stalk being the ligament of attachment. The general structure posterior to the pyriform bodies is granular, the various sized granules uniting, as development proceeds, into sarcous masses, which are separately movable. There are contractile bodies in pairs, one pair lying behind and between the pyriform bodies and the stomach, on a level with the anterior coronal apertures, the other pair placed on a level with the posterior coronal apertures. They form part of a complete water-vascular system, and pulsate vigorously at the rate of 240 beats a minute. Viewed laterally they form short tubes, 7 μ in diameter, with well-defined walls, in which a fluid can be seen circulating rapidly. Looking at the vessel longitudinally, there is in its lumen a most curious dark tongue-shaped central object, which moves at the pulse rate with a peristaltic undulation from base to apex, the shape being retained in spite of the movements.

The anterior bodies are directed up and back, the posterior are more transverse, and I have traced the latter to within a short distance of each other across the body, but they are always lost eventually beneath the mass of granules in which they are embedded. The arrangement of the vessels into cephalic and caudal circles (Plate III), with longitudinal anastomoses, is only to be seen in the most mature ova. Brock has described them as having communications with the posterior coronal apertures, and has succeeded in tracing the vessels directly, thus accounting for the power of the embryo to contract and expand so markedly during its struggles to free itself.

Under observation the cilia are seen in constant motion, while the embryo itself moves in an unpleasant, spasmodic way—the sarcode masses being drawn in a semi-peristaltic fashion toward the head end. The spine of the ovum is the relatively fixed point, and the proboscis is the fulcrum by which attempts are made to tear away the ligaments attaching the shoulder to the lining membrane

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—the slipping of the head, as the effort fails, looking as if the snout were searching for or scraping an opening. Cobbold's account is so graphic I cannot do better than quote it:—"When the ciliated animalcule has succeeded in overcoming this first difficulty (tearing the ligaments) it is ludicrous to witness its frantic efforts to find an opening in the shell. While thus partially liberated it will rush to and from one pole of the egg to the other, performing a series of summersaults, and at the same time occasionally rolling itself over laterally. This activity becomes gradually more and more violent, until at length its excitement is worked up to a sort of frenzy." In addition to this there is marked increase and decrease in the size of the embryo, which can be best explained by the expression of the fluid through the coronal apertures from the water-vascular system—an hypothesis which possibly also explains the presence of some of the granules lying free in the peri-embryonic space. At length the efforts are crowned with success, the shell bursts longitudinally in two-thirds its length, and the embryo escapes head first, often darting with great velocity out of the field of the microscope, but mostly pausing in an hour-glass shape for an interval. If it be incompletely freed it dashes about shaking the attached shell in its violent struggles. Usually the lining membrane is left *in situ*, but it may be extruded to lie as a rounded mass beside the empty shell; more rarely it is freed from the shell, but attached to the embryo, which swims about with its cilia, apparently not minding an impediment which streams behind it, and makes but little difference to its speed.

When swimming the speed is so great that it can only be watched with the low power of the microscope, and the movements are most dog-like: it seems endowed with actual intelligence as it moves briskly about, never resting, examining every obstacle with its snout, avoiding larger obstacles, pushing aside the corpuscles, passing in and out under the edge of the cover-slip with the eagerness of a dog. When resting it assumes a rounded form, but this is rare, and in plain water it seldom lives more than 24 hours, the movements getting less active until it assumes the circular form and dies—the cilia at the shoulder ceasing to move last, and the contractile bodies continuing to beat after all the cilia have ceased to move, but growing slowly feebler and feebler. The sarcous masses draw up convulsively less frequently, and stop wholly before the death of the cilia. As soon as the pulse is gone the body begins

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to swell by the entrance of water, and speedily all trace of structure is lost in a granular degeneration. There are various modifications of the typical round dying form varying with the cause of death; apparently in any poisonous medium the head is retracted, while an unusually clear micro-photograph shows the characteristic appearance of the empty shells (Plate IV).

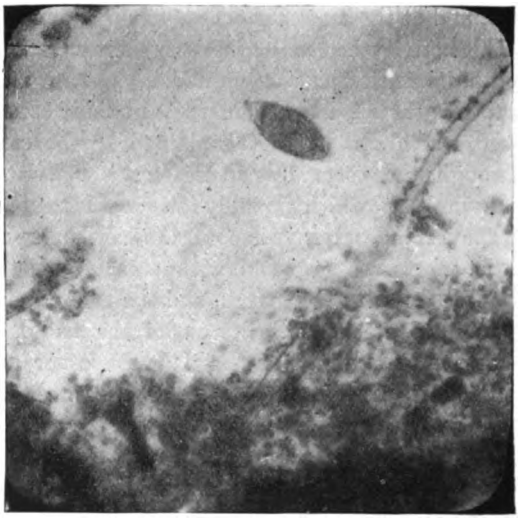

PLATE IV.
Micro-photograph, showing the empty egg-case.

Having described the usual appearances, I will make various observations which had better be given in note form:—

(1) With the centrifugal machine the ova are always demonstrable, often months after the cessation of hæmaturia has led the patient to think himself cured, and such ova are usually well developed.

(2) The anatomy is best studied in neutral or faintly acid urine, and often dilution is necessary before hatching occurs; but, contrary to emphatically expressed belief, dilution is not absolutely necessary, as I have watched embryos hatch out in urine when undiluted, but of low specific gravity and of almost neutral reaction—this being a fact which the constant presence of empty shells had long led me to suspect.

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(3) Effects of reagents:—HCl has fatal results 1 in 2,000 (one-fourth the amount of HCl present in gastric juice), killing in half a minute, the embryo becoming round and motionless with dramatic suddenness as the acid reaches it in its active movements. KHO, 1 in 2,000, kills as surely in double the time; methylene blue in aqueous solution is fatal in from three to five minutes—that death was directly due to its effects was shown by the fact that the embryos in the central area of the slide, where less of the dye had penetrated, were the last to die; benzoic acid, 1 in 1,000, killed in 10 to 15 minutes; carmine, $KMnO_4$, and alcohol, 2 per cent., are all fatal, and most of these toxic agents, if not all, produce retraction of the head before death. The least decomposition is sufficient to prevent the hatching of ova, which, however, will hatch in sea water.

(4) Passed into pure water the embryos are hatched in thousands in a short time—free dilution of the urine with aqua destillata enabling the process to be observed within two minutes.

(5) Effect of blood.—I have with me a slide of ova kept in blood serum for 24 hours at 30° C., and in it is an embryo near an empty shell, from which it hatched under my direct observation. I have this same embryo now under the microscope, and shall be pleased to demonstrate it at the close of the meeting. This is a most interesting and, I believe, quite new success, with important bearings upon the question of auto-infection. Besides the one embryo hatched out, sluggish movements were to be seen in various other ova after 24 hours.

Development.

As having important bearings on the development of Bilharzia, I may be permitted perhaps to run over the stages of reproduction of the allied liver-fluke—the type of digenetic trematode development, as shown in Plate V:—

(1) The operculate egg containing the embryo.

(2) The "miracidium," or hatched larva, is very like that of Bilharzia, swimming freely with cilia, and possessing eyes, brain, and stomach.

(3) The larva bores its way into a fresh-water snail, loses its cilia encysts, and forms a "sporocyst" containing asexually-formed "rediæ" (x) *viâ* a gastrula stage (xxx) and "germ-balls" (xx the latter of which may contain a second generation of rediæ.

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(4) The redia escapes from the parent embryo, moving partly by the aid of [two rudimentary locomotory processes (x). Having found a convenient locality it produces the "cercaria," and is seen to contain more germ-balls, which may themselves contain a third generation of rediæ. The cercariæ are born singly through the birth-pore at (B).

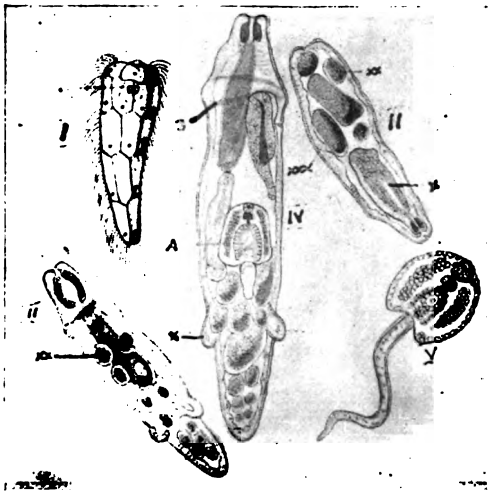

PLATE V.

Showing the stages of development of distoma hepaticum :—(I) Miracidium with ciliated epithelium ; (II) Sporocyst with rediæ (x), gastrula stage (xxx), germ-balls (xx) ; (III) Redia with rudimentary locomotory processes ; (IV) (A) Redia with cercaria, (B) Birthpore ; (V) Cercaria.

(5) They possess in a rudimentary form all the organs of an adult fluke—suckers, pharynx, stomach, eyes, and genital organs. By its vigorous tail the cercaria escapes from the body of its host, swimming freely for a time, then it adheres by its sucker to a grass blade, loses its tail, encysts itself in a mucoid material, is eaten by a sheep, freed in the stomach, and creeps up the bile-duct as a young fluke to complete the cycle by ovulation.

The diagram of a bucephalus cercaria from the liver of an anadon shows graphically the difficulties of research arising from the various cercariæ of this group, being so unlike either their parents or each other. The difficulties presented by Bilharzia are increased

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by the fact that it is an exception to the almost universal rule that the intermediate host of this class is a mollusc—Sonsino, in 1893, demonstrating the exception after two years' work, of which the following is a brief *résumé*:—

There are at least two forms of intermediate host:—(1) A small (8x3mm) arthropod—the crustacean amphypoda, which is invaded by the embryo encysting in the first segment near the eye. The cyst is globular and 120μ in diameter; it contains the embryo, which can be seen to move when warm water is run under the cover glass, the only difference from other distoma sporocysts being the characteristic beading of the integument, as it is seen in the normal mature embryo. It has lost its cilia and gained two suckers, whence its name, "Dicotyle," while a pair of ciliated infundibula are developed, a pair at each terminal third. Any forcible rupture of the cyst always kills the embryo. The host is ingested in drinking water, the cyst is dissolved in the stomach, the liberated embryo holds on by its suckers to the intestinal mucous membrane until it works its way through by means of its proboscis; having thus reached the lumen of a small venule it metamorphoses into an adult worm by stages that at present can only be imagined. Zoologically its interest lies in the fact that it undergoes no alternation of generation, and passes through no redial stage of asexual reproduction as do the typical distomes, but resembles the holostomes in that the ultimate encysted form is a direct encystation of the original embryo, and not of an intermediate cercaria. The Bilharzia develops neither by alternation of generation nor by asexually formed rediæ, but by simple metamorphosis—by "metastasis" as Leuchart termed it. Grobben suggests that the germ-balls are essentially ova contained in the rediæ, and that the cercariæ thence derived are no more asexual than the rediæ themselves—but that is a question more interesting than relevant to the scope of this paper.

(2) The other intermediate host is the larva of an aquatic insect, the ephemeridæ of the same size as the amphypoda. Cobbold's belief that a gasteropod can act as host is incorrect, and no mollusc has yet been found to act in this capacity.

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PATHOLOGY.

The Bilharzia embryo has been already traced as far as the radicle of the portal vein, which, from its parasitic instincts, it penetrates as a convenient position for obtaining food. The entrance causes probably ecchymosis and stasis, the latter providing an incentive to proceed along the lumen of the vessel until the blood-current is met with in a larger branch. As metamorphosis and development proceed it is obvious that the successive obstruction of larger and larger branches causes migration until, with maturity, a considerable vessel is reached—one large enough to allow of the presence of the two sexes in apposition. As the number of worms present has been supposed to bear relation to the number of sporocysts ingested, it is not surprising that no less than 300 have been found in the portal system in one autopsy.

Having developed, the worms live in pairs, the female lying in the gynæcophoric canal, and never being found alone, save when encysted to ovulate. The oviducts are crowded with ova—fertilization occurring apparently by direct penile intromission, the apposition of the genital pores being maintained by the folding around of the lateral margins of the male. The spermatozoa are, as has been shown, aspirated through the oviduct, after being lodged in the antrum femininum, by the action of the cilia in Laurer's canal, which leads from the commencement of the oviduct to the dorsal surface. The male is enabled to proceed against the blood-current by the tuberculation of the dermis, while the female, being smooth, is not, hence she is borne by the male down the vein until it becomes too small to admit of his further progress, when it is small enough to afford the female a hold, and she proceeds alone until arrested by the small size of the vein, when she encysts and ovulates in the lumen. The smaller radicles of the vesical veins are the most affected, and the hæmorrhoidal next so, but other localities are involved which are overlooked, as they afford no clinical signs of the invasion. The cysts—which may reach the size of a lentil—are scattered in the bladder wall, the tension in them increasing as the individual ova reach maturity, until it is so great that rupture is produced by the contraction of the muscular coat—a fact shown by the blood flowing only at the end of micturition, when the powerful muscular coat has the greatest purchase, and by the signs of basal cystitis in the area in which that muscle is most developed.

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The following notes of an autopsy by Curston in 'The Lancet,' in 1889, show that other radicles may be affected :—

"Patient was stationed in Maritzburg 12 years before death, which resulted from heart disease. On removing the intestine nothing was noticed until the large size of the appendix arrested attention. It was twice the usual size, distended at its extremity, and felt gritty; it was non-adherent and yellowish at the extreme end. Several hard nodules projected slightly beneath the peritoneal surface, some as large as mustard seeds. On section each nodule was found to consist of several areas of dense connective tissue surrounding bodies lying in groups of from three to 20; these bodies microscopically were oval, with a spine at one end about ·06 mm. in length. In some cases the capsule only remained, being shrivelled and unrecognizable, only an occasional one still retaining the characteristic shape of Bilharzia ova, making it possible to be sure that it was a case of old standing distoma disease." In this case, there being no adhesions indicative of old peritonitis, there would have been probably no signs other than the passage of blood either overlooked or attributed to dysentery.

Sonsino has an elaborate classification of the effects on the bladder wall, which at least serves the purpose of preventing this disease from being taken too lightly:—(1) Infarctive; (2) Hæmorrhagic; (3) Granulative—with thick soft granulations, the result of irritation in the inter-papillary spaces by retained mucus and ova; (4) Incrustation—by calcium salts, crystals of uric acid, and ova; (5) Ulceration, which has been known to go on to perforation of the peritoneal covering; (6) Vegetative—the fibro-adenomatous masses being pedunculated, though rarely reaching a size calling for surgical treatment; (7) Pigmentation.

The ova may give rise to calculi, of which they or the salts of phosphatic urine to which they give rise, form the nuclei. The kidney is enlarged, congested, even showing ecchymoses or distinct hæmorrhages. The lining membrane is affected similarly to the bladder and ureter, but to a less degree. I can trace no record of the worm having been found elsewhere than in the portal radicles, but the ova have been met with in almost every tissue, as would be expected, seeing that they have been discovered in the vena cava and the cardiac ventricle. The few ova that escape—causing non-infective emboli—are unlikely to cause serious trouble.

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I have at present an interesting ophthalmoscopic case in which the question of multiple embolic hæmorrhages arises.

Death is usually the result of pneumonia, which the effect of the active poison secreted by the trematodes renders the patient less able to resist. It is a rare occurrence in any case. Other causes cited are Pyelitis, Pylephlebitis, Pyonephrosis, Hydronephrosis, perforation of the bladder-wall and pseudo-dysentery with hepatic abscess—the last of which I have already called attention to. The liver is, however, very rarely affected—a curious fact when the portal vein is the seat of the disease.

To consider the bearing of these various data:—First, concerning the channel of invasion—mouth *versus* urethra. The areas involved being those of the intestinal and urinary tracts, it has been inferred that the parasite has made its way in by the apertures leading to these cavities while the patient has been bathing, it being urged that the localization to these tracts thus laid open is too definite to admit of other explanation. Anatomically considered, however, it will be seen that the distribution and anastomoses of the portal vein afford an equally accurate explanation. The vesical branches—the hæmorrhoidal anastomoses and the mesenteric radicles—are all affected, and no other lesion has been found which cannot be met by a consideration of the eight peripheral anastomoses of the portal with the systemic veins, and the realisation of the fact that chance ova are passing from the oviducts of the female into the blood-current before she has encysted for ovulation. The presence of renal and ureteric affection, cited as evidence of invasion from below, is explicable by the renal and sigmoid-ureteric communications which exist. It is to be asked, Why, when the anus affords so much readier an entrance, should the vesical cases so remarkably preponderate? It is not difficult to see why this is so on the rival hypothesis, but the fossa preputialis seems an inadequate explanation of this difference. Another point noticed is the remarkable preponderance in males, said to be due to their almost living in the water in the summer as boys. It is stated that in some parts of Natal hæmaturia in boys is so common as to be looked upon as physiological, and that in the Fellaheen in Egypt no less than 75 per cent. of the men are estimated to be affected, while Brock states that in his immense experience he has hardly met with a case occurring in a female.

These facts are very significant, but it must be remembered that

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hæmaturia in white women is not likely to attract attention unless it be excessive, while in the natives no one who has seen their women fording rivers can fail to realise the risk of urethral invasion. It may be urged that if Europeans of that sex do not bathe, the claims of the bath are not so wholly neglected as to eliminate the chance of infection. Sonsino's researches are important evidence, but unless there be some protective changes developed while in the sporocyst the power of the embryo to live after liberation is queried by my observation that HCl, two per 1,000—(the acidity of the gastric juice)—is fatal to it in less than two minutes. And there seems to be no means of explaining the preponderance of cases occurring in males. The diameter of the female being only 1-90th inch, the veins she reaches are very small, and as statis and encystation occur the hæmaturia proceeds from the ruptured mucous membrane, which yields to the pressure resulting from development of the vast number of individual ova. It has been said that "the life of an entozoan is equivalent to that of its host," but it is unlikely that ovulation should be delayed so long as 15 or more years under continuously favourable conditions. If no intermediate host be required it is possible that auto-infection as suggested by Sonsino may occur, new generations being thus produced, and in this connection lies the interest of my observation that the embryo will not only hatch and live in urine, but also in blood-serum.

A recent writer says: "Sonsino's theory of auto-infection—in other words, that the animal once lodged in its human host is capable of completing its life cycle therein—may be confidently rejected, not only on account of its zoological improbability, but from the fact that the embryo will neither hatch nor live in urine or blood." Of the "zoological improbability" I cannot express an opinion, but that the embryo can hatch both in urine and blood I have succeeded in proving experimentally, with confirmation of both results. It is to be remembered that it is the identical embryo which is originally hatched from the ovum which enters the sporocyst, to be freed eventually in the alimentary tract of the host; and if the metamorphosis can occur after liberation and invasion of the portal vein, it is reasonable to suppose that it may also be effected in the portal vein direct. Dead females are found in the vesical cysts, and in other cases it is not easy to see by what means they are to effect an escape from the enclosing fibrous tissue surrounding the cyst. Were each female to die after ovula-

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tion, if there were not fresh infection going on, the disease must wear itself out unless the number of worms were immense or auto-infection occurred. Cases go on almost indefinitely without reference to fresh infection from without, some having been known to recur after 18 years, the patient supposing himself cured long since. The number of worms inhabiting one portal system cannot be legion, so that there are other reasons in favour of the auto-infection theory that are plausible. Prophylaxis should be easy if Sonsino's work be accepted, for the intermediate host is in either case from one-twelfth to one-fourth of an inch long, and hence easily removed by filtration through even coarse linen. The medium most favourable to the life of the crustacean host is standing or slowly running water with sedgy banks, such as is found in the veldt pools after the rains. The temptation of these pools to our men on a long, dry, dusty march was irresistible, and the absence of fuel made boiling impossible. True, filtration through a handkerchief would be efficient, but from my experience of handkerchiefs on the veldt I am not enthusiastic *re* their use as filters of water for personal use.

It will be noticed that both intermediate hosts described are aquatic. Geographically the disease is found scattered all over Africa, and cases are reported from Arabia, India, and Madagascar. All my cases traced their infection to the Rustenberg district of the Transvaal, save one from the borders of the N'Gome forest, but it is impossible to determine the source of infection when the patient has been trekking all over the country for months.

Clinically.

The incubation period is very constantly from four to six months between exposure to infection and the onset of symptoms, of which slight vesical irritation is usually the first, to be followed in a few days by hæmaturia, the dysuria then increasing rapidly. Any attempt to retain urine is accompanied by severe pain localised in the perinæum, not relieved by micturition, and most intense when, as often happens, a small clot is impacted in the urethra. The urine flows quite clear at first, to be followed by blood, more or less pure, at first a few drops only, increasing to an average of a drachm at each micturition, the amount passed thus depending upon the amount of vesical irritation. Commonly, the acuteness

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increases until the frequency of emptying the bladder is every half-hour, and then the condition becomes steadily less acute as rest is enforced, until only a few drops of blood pass, when the condition remains stationary for months, the amount passed varying as the amount of exercise taken. If the patient be removed from the affected district promptly after exposure to infection, and the number of embryos ingested be small, the subsequent trouble is little, the slight hæmaturia alone remaining subject to exacerbations on exertion. The anæmia is soon removed under treatment, and the patients show no marked depression. On pressure on the trigone per rectum there is usually marked tenderness. There is this curious fact, that the hæmaturia is worst in the morning from 9 a.m. to 2 p.m., and it may entirely cease during the afternoon. The urine is of specific gravity 1015–1025, usually acid, unless alkaline from cystitis, and albuminuric in proportion to the hæmaturia. In it various pathognomonic filiform bodies have been described, and chyluria has been observed occasionally. Casts are constantly found of mucus, which forms in the crypts, and being forced out by pressure from below breaks off with embedded ova, and is passed in pieces of considerable size. The amount of the daily total of urine passed is not increased to any great degree. In the rectal cases the symptoms are those of sub-acute colitis, ova being found in the mucus.

The Blood.—Under the microscope the corpuscles show no change, and the great excess of leucocytes seen in the hæmaturic urine is due to the accompanying cystitis. In a series of cases examined the average per square in a Gowers' hæmocytometer was 45, while by Thoma-Zeiss's hæmoglobinometer the percentage was estimated at 89. These cases were chronic convalescents, and the figures show that the anæmia is persistent.

Opthalmoscopically.—Examination of all cases systematically produced no results until I found a case of multiple hæmorrhages, on which Major Yarr, R.A.M.C., has kindly given me the following notes:—"The right fundus appears to be normal. In the left fundus are several small hæmorrhages, apparently of some standing, on and around the terminal branches of the superior and inferior temporal arteries. There is little pigmentary disturbance, the macula is unaffected, the disc is clear, and the vessels are of normal calibre. The opthalmoscopic picture is quite unlike that of the usual form of albuminuric retinitis. Vision is unaffected, save for the scotomata due to the hæmorrhages."

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THERAPEUTICALLY.

There is not much to be said on this aspect. The situation of the parasite in the blood stream makes it very improbable that there will ever be found any means of eradicating it from the system. But much can be done to lessen the symptoms of hæmaturia and cystitis, and to combat the subsequent anæmia which is the patient's main trouble. First comes methylene blue as the most useful drug yet used. Given in four-grain doses three times a day it has most satisfactory results. It appears in the urine in 15 minutes after administration, causing an increase in the amount of vesical irritation for 24 hours; then there is a marked lessening of the hæmaturia, which is maintained for five or more days. In one patient, who had had incessant hæmaturia for three months, passing 2 ozs. of blood a day on an average, there was a distinct diminution after two doses, and an almost complete cessation after the fifth administration, lasting for two days. Experimenting with the drug, it was found that when a loop of fowl's gut containing it was suspended in a tube of diluted urine, the blue passed the membrane speedily and was absorbed by the ova, some of which were deeply stained and others not at all; the stained ova showing no decrease of activity, and hatching as usual, but the embryo died in less than 15 minutes on the stain being run under the cover glass. On dying the ova that were stained discharged the blue, those that were not stained showing no activity whatever. The affinity of living tissue for methylene blue is shown by the staining of all the organs if toxic doses (6 grains) be given, and that the urinary system is particularly affected is shown by an autopsy in a case in which it had been administered, the peritoneal coat even of the bladder being deeply stained. That it has powerful microbicide action has long been known, Dr. Austin Flint having reported a cure, by its exhibition, of a case of chyluria from filaria sanguinis hominis in the 'New York Journal' of 1895, and Dr. Smith, of the United States Navy, having used it successfully in malarial hæmaturia, but it was objected that the good results were only apparent, and that the effect produced was not due to the actual lessening of the blood passed as such, but to the formation of the innocuous but stable methæmoglobin. However this may be when given in toxic doses, it is not so in cases on the quantity suggested, for the spectroscope showed no

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trace of the three-banded spectrum in either blood or urine in a case in which it had been given for some time, the urine being of a deep blue. It is fairly certain from the data given that the blue is taken up by the ova in the oviduct, and that it exercises an inhibiting effect on the immature ova in their development is my opinion. It does not affect the mature ova, but it has again a marked effect on the embryo when hatched.

Concerning administration, it is perhaps worth noticing that if the abbreviation "Methyl. blue" be used there will be given the toxic preparation of that commercial name with unpleasant results; and in cases in which ill effects result from the exhibition, it is as well to remember that with caustic soda the methy*lene* blue turns violet, and the methyl blue turns red. It is best given in four-grain doses three times a day, with a little hyoscyamus to lessen the vesical irritation, or better, perhaps, with some powdered nutmeg. It should not be given for more than two days at a time, as the lessening of the hæmaturia is maintained for from five days to a week after the one day's administration, as a rule. The drug most appreciated by the men themselves is benzoic acid, for which they ask if it be discontinued, on the plea that it reduces the irritability of the bladder. The cause of its being given at first was the observation that the embryo hatches less readily in acid urine, and that it cannot live for 15 minutes if a solution of 1 in 1000 be run under the cover glass. I have, therefore, given it in the intervals of administering the methylene blue on every fifth day, and have combined with it hyoscyamus in fairly full doses. The action of benzoic is more powerfully antiseptic than that of carbolic acid: the administration of the latter is attended with, in my experience, no good results. It was given a trial in the form recommended—viz., the glycerinum acidi carbolici. The secret of the success attending the use of benzoic acid may lie in its action in lessening the reflex irritability of the bladder, or in the fact that the amount given *per os* is passed in almost its entirety in the urine, as hippurates with the ammonium present when cystitis exists, thus diminishing the alkalinity due to triple phosphates. Urotropine has been well tried. Being a compound of ammonia and formaldehyde, it was thought that it formed formaldehyde in the urine, and that it might thus attack the parasite *en route*. Although this theory of the liberation of formaldehyde has been disproved, the action of the drug in cases which are complicated by cystitis of a more

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acute type than usual is most useful as a powerful urinary antiseptic in 15-grain doses. It has, however, no specific action, only affecting the cystitis, which complicates the disease. Suprarenal extract has been recommended as arresting the existing hæmaturia, but I have seen little benefit from its use in five cases to whom it was administered for a week—·5 grain eight-hourly has an immediate effect, but toleration is soon established. Terebene is said to be useful in cases of cystitis, and has been given in half-drachm doses night and morning. The tenesmus accompanying the rectal cases is relieved by injection of plain, warm boric lotion, but it is not to be expected that any cure will be so achieved. It is in those cases with much discharge that liver abscess is to be most feared.

In conclusion, it may be said that treatment is symptomatic and empirical, and that no permanent check can be placed upon the disease unless the most careful observation of healthy conditions be maintained, but that if due care be exercised the hæmaturia can be so diminished as to be almost harmless, and the patient's condition one that is surely satisfactory when the nature of his malady is considered. Finally, I wish to express my thanks, Mr. President, for the opportunity of reading this paper to-night, for the courteous reception you have afforded it, and in closing I should like to express my indebtedness to Colonel James, R.A.M.C., for his able assistance in the preparation of my figures and plates.

Surgeon-General Keogh thought it would have been well if the author had dealt less with zoology and had entered more fully into treatment and clinical symptoms. The subject would very shortly be of great importance in this country, and not simply of interest to those in practice in Africa. There would soon be a large number of cases of Bilharzia in England, and at the present time many were being treated in the various hospitals. In this country it was often mistaken for dysentery or for leucocytosis and allied conditions. Mr. Lelean's paper was a most opportune and useful contribution to our knowledge of a disease which, though not often seen in this country, was quite common in South Africa.

Colonel W. L. Gubbins bore testimony to the importance of the subject. He had been principal medical officer at Pretoria during 13 months, and there saw a large number of cases of Bilharzia, fully 90 per cent. of them having come from the Rustenburg district, in the north-west part of the Transvaal. During the last three months a large number of the invalids from South Africa suffered from the condition, and the majority of them also came from the same district, or from the borders of Swaziland, where, during General French's sweeping movement, something like 10,000 men had been engaged. He had not had time during his busy life to go into the pathology and treatment of the disease, but he regarded the subject as one of very great interest.

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Dr. Bryant said he had had three cases under his care, one of which came from Rustenburg, another from Crocodile River, and another from the Zambesi. The first two traced the condition to bathing; the third did not bathe in the river, but the water composing his tub was drawn from it, therefore the case was practically on the same footing. He was glad to hear the suggestion for the treatment of such cases. His patients had been on urotropine, but with no great success. The case from the Zambesi was a mild one, and it did very well on glycerinum acidi carbolici. The hæmaturia disappeared, and the patient left Guy's Hospital in three weeks, neither blood nor ova having been in the urine for several days. One of the other men developed a curious complication. He had been treated by washing out the bladder, and a short time before coming to Guy's Hospital he developed rigors and a high temperature, so that malaria was thought of. Examination of the blood showed no malaria parasites, but there was a pure culture of streptococci. It seemed that the man had septicæmia resulting from cystitis, which was probably due to streptococcal infection. The chief point in the treatment brought out in Mr. Lelean's paper appeared to be the administration of methylene blue.

The President thought the Society was to be congratulated on having such a paper presented to it, and he felt sure the Fellows would extend to Mr. Lelean their very grateful thanks for the trouble he had taken in preparing so interesting a contribution. It was very important to be forewarned and forearmed in the way they had been, and particularly as many of the Fellows probably had no more than a book knowledge of the condition. It was most gratifying to have placed in one's hands a weapon with which to combat a malady which would probably be invading the country. It was also extremely satisfactory to find that work of the character represented by the paper was being carried on by the officers of the Army Medical Department under the auspices of the chiefs of that department. That was to be regarded as of valuable promise for the future.

Lieutenant Lelean briefly acknowledged the cordial thanks which had been tendered him, and regretted that lack of time had compelled him to omit portions of the paper. He concluded by exhibiting on the screen a series of micro-photographs.

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May 12th, 1902.

THE DIAGNOSIS OF MALARIA FROM THE STANDPOINT OF THE PRACTITIONER IN ENGLAND.

By PATRICK MANSON, M.D., F.R.C.P., C.M.G., F.R.S.,
Fothergillian Medallist, 1902.

SLIPSHOD diagnosis is not to be commended. We must confess, however, that, so far as the interests of patients are concerned, in the vast majority of diseases, provided the physician abstain from a too active interference with natural processes, it matters little whether diagnosis be exact or even correct. In too many diseases we possess little or no power for good. Fortunately for the reputation of our art there are a few diseases over which we can exercise absolute or relative control; and just because we possess in the case of these remediable diseases this power, it is of the utmost importance that we recognise them, that we diagnose them accurately. The disease about the diagnosis of which I propose to offer a few remarks this evening—malaria—is one of these. For it, in quinine, we possess an almost infallible remedy. Hence, if we fail to recognise malaria when it is present, and in consequence of such failure in diagnosis omit to administer the appropriate remedy, the patient suffers, and we grossly fail in duty. Contrariwise, if in some given case we erroneously diagnose malaria and treat the patient accordingly, we certainly treat the case wrongly, perhaps miss the chance of cure by more appropriate means, waste a valuable drug, and certainly run the risk of damaging our own reputation. The ability to diagnose malaria is, therefore, a matter of high practical importance.

In those countries in which malaria is endemic and common, it not infrequently happens that the infection is overlooked. As a rule, however, it is the other way about; malaria is far too frequently assumed to be present where it has nothing whatever to do with the case. This remark is equally, if not more, applicable to certain cases of disease which come to England from abroad. Such is the exaggerated idea of the insalubrity of tropical countries, of the universal prevalence of malaria in them, that a very large

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proportion of the febrile, and even non-febrile, diseases coming from them to this country are diagnosed and treated as malarial. It happens far too often, both in England and in the tropics, that the practitioner, when in difficulties about a tropical patient, assumes the case to be malarial. The patients, likewise, are very prone to be content with and to endorse such a diagnosis, for they too, from one cause or another, have come to believe that in the matter of disease "tropics" spells "malaria." Somehow to many there seems to be a sort of fascination and appropriateness about the word "malaria," as if it carried with it mystery, romance, and explanation, such as is said to belong to the blessed word "Mesopotamia." In the course of 12 years' experience of London practice I have seen some two or three cases only in which malarial infection had been overlooked; in the same time I have seen a very large number of cases in which malaria had been diagnosed, but in which malaria was neither present nor had anything whatever to do with the symptoms.

The latter type of case I meet with so frequently that I thought, when asked to read a paper before this Society, I could not do better than choose for my subject this very practical matter of the diagnosis of malaria as we find it when imported into England.

Whatever may have been the difficulties in the way of reliable diagnosis of the infection in the past, recent discovery has enabled us to be precise and absolutely sure about it, at all events in 99 cases out of a 100. There are few diseases so easily diagnosed; nevertheless, and although nearly every member of the profession knows, in an academic sort of way, the principles on which the points of diagnosis are founded, my experience tells me that these principles are not applied systematically, nor in actual practice assessed at their proper value. Possibly it may serve some useful purpose if, availing myself of this opportunity, I recapitulate and accentuate these principles.

The pathognomonic indications of malarial infection may be conveniently arranged under these headings:—(1) The clinical; (2) the therapeutic; (3) the microscopical.

(1) *The Clinical Diagnosis.*—There are many signs and symptoms of malarial infection—fever, anæmia, icteric tinting of skin and scleræ, enlargement of the spleen and liver, gastro-intestinal disturbance, a tendency to spontaneous recovery and to subsequent recurrence. All or any of these may lead one to suspect, but not

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one of them, nor the concurrence of all of them, justifies a positive diagnosis of malaria. None of them, and no grouping of them, is pathognomonic. They are present separately or variously combined in many other morbid conditions. They may justify in any given case a suspicion, but not a diagnosis, of malaria.

There is only one clinical symptom that justifies a positive diagnosis: this is periodicity, periodicity of a particular type—tertian or quartan periodicity—a rhythmical recurrence of symptoms every 48 or 72 hours. In no other morbid condition are these to be found, and wherever well marked malaria is present.

In malarial disease the appearance of quotidian periodicity is common enough; but it is only the appearance. It is very doubtful if true quotidian periodicity ever occurs in malaria; if it does it is certainly very rare. The parasites of malaria are either tertian or quartan in habit, and many of the symptoms they give rise to are correspondingly tertian or quartan in their recurrences. Multiple infection by two kinds of parasites, or by two or three generations of the same species of parasite, may give rise to double tertian or treble quartan infection with diurnal recurrence of symptoms; but this pathologically, whatever it may be clinically, is not a true quotidian periodicity. It is common enough in malaria, but I would insist it is not pathognomonic of malaria, for a similar quotidian periodicity is very general—in fact, is usual—in almost all febrile complaints. Therefore, to make quotidian periodicity a ground for a diagnosis of malaria is wrong. More than that, it is most dangerous, and is, perhaps, the commonest cause of erroneous diagnosis of malaria. Experience has made me formulate a rule for myself which I commend to you—have absolute confidence in tertian and quartan periodicity as a basis for a diagnosis of malarial infection, but distrust, or rather absolutely disregard, quotidian periodicity.

In tropical practice I have seen more mistakes from relying on quotidian periodicity as a ground for diagnosing malaria than from any other cause. The temptation to attach weight to it is often very strong, but I would again urge on you to harden your hearts against it.

When a patient from the tropics comes to you with a story of evening rigor, rise of temperature to 103° or perhaps 104°, and a profuse sweat, the idea of malaria naturally presents itself. Most probably the patient tells you he has malaria. And if one

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finds the patient anæmic, and that his spleen is large, perhaps very large, the diagnosis of malaria seems to be clenched. But people abroad get splenic and spleno-medullary leucocythæmia, and other diseases in which the spleen is enlarged, as well as people in England, and such patients often have marked quotidian fever. These leucocythæmic and other kinds of enlarged spleen cases I frequently see on the supposition that they are malarial. Had the patients never been abroad, probably the idea of malaria would never have been entertained.

Another type of case in which there is enlargement of the spleen associated with anæmia and quotidian fever I occasionally see, probably because it, too, is sometimes supposed to be malarial. In a sense the cases I refer to are malarial, or, rather, post-malarial. I allude to what is known in Assam—where the condition in certain districts is very common and very fatal—as Kala-azar. The same condition occurs in other malarious countries, however. The patient gives a history of malarial attacks, which at first were controlled to some extent by quinine. After a time the recurring attacks became more frequent and less amenable to quinine. Finally, the fever resisted treatment, became quotidian, the spleen becoming permanently enlarged, the anæmia intense, the skin sallow or even melasmic, and, ultimately, in spite of treatment, the patient, if not cut off by some intercurrent disease, dies of pure anæmia. This disease, although possibly induced by malaria, has long ceased to be malaria—is, in fact, a post-malarial cachexia, brought about by organic changes in the blood organs, and induced possibly by some additional and distinct infection attacking organs already damaged by antecedent malaria.

I find that the presence of enlarged spleen is a bait that few resist when attempting the diagnosis of a quotidian fever in a patient from the tropics. The combination is so suggestive. But we must bear in mind that many people in the more malarious districts in the tropics have, though free from active malarial disease, chronically enlarged spleen; and we must also bear in mind that the possession of an enlarged spleen is no protection against other diseases, and that enlarged spleen is symptomatic of many diseases, febrile and other. I have several times seen ulcerative endocarditis in patients with spleens enlarged from antecedent malaria; a coincidence merely. The same occurrence I have often seen in phthisis, and in many septic conditions.

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If quotidian fever associated with enlargement of the spleen is to be distrusted as a combination indicative of malaria, still more so is quotidian fever associated with enlargement of the liver. The latter combination usually means abscess of the liver. Any one who has had much experience of tropical practice must know that this disease is often, I might say generally, at all events in the earlier stages, diagnosed malarial fever. Osler says he has seldom seen an abscess of the liver in which the patient had not been liberally dosed with quinine. This is my experience likewise. I have seen a medical man make the mistake in his own person. The gentleman I refer to had been many years in India. He fell ill, and was invalided home. He had every afternoon a smart shivering fit, a rise of temperature to 103° or over, followed by drenching perspiration. He took large doses of quinine daily for many weeks. Finally, becoming very ill, he sent for me. I found the liver enlarged, tender, the spleen not enlarged, or only slightly; these facts, with the quotidian fever and other points, satisfied me that there was an abscess in the liver. But I could not convince the patient. He asked me to make a blood examination. I did so, after suspending the use of quinine for a week to give the hypothetical plasmodium a chance to get into the peripheral circulation. The result, so far as the presence of the parasite was concerned, was negative. Next, he insisted on another week of larger doses of quinine, and only then, and after much persuasion, did he accept my diagnosis and permit Mr. Godlee to open the large abscess in his liver. Fortunately he got well, but his conversion to the fallibility of quotidian periodicity as a diagnostic mark of malaria was somewhat dearly purchased.

Strange to say, syphilis, both secondary and tertiary, is not infrequently, on the strength of an associated quotidian fever and a residence or visit to a malarious country, diagnosed malaria. Some years ago I was consulted by a gentleman who had resided for many years in a mildly malarious country. The story he told me was that for over a year he had been suffering from ague. The attacks, he said, were quotidian and very severe. He had taken large doses of quinine and of arsenic, had visited hydropathic and similar health establishments, and done everything he could think of to get well. As soon as he told me the story of his treatment, for reasons which I will state by-and-by, I was quite sure he was not suffering from malaria, and proceeded to endeavour to ascertain

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the cause and nature of his fever. In the course of my examination I found his liver very much enlarged, studded with irregular firm masses so big and hard that I thought they were probably malignant. I asked if any one had examined his liver before. He told me no one had done so recently; everyone had accepted his own diagnosis of his case (a very common practice, I am sorry to say), for, said he, of course he knew all about the symptoms of fever much better than any doctor, in this country at all events, could tell him. In going further into his history I ascertained that many years ago he had a chancre. In the hope, but I confess not with the expectation, that the lumps on his liver were syphilitic and his fever symptomatic of softening gummata, I put him on iodide of potassium. Two days later he called on me saying the medicine had given him headache and coryza, and so upset him that he had stopped taking it. After a good deal of trouble I persuaded him to double the dose. A week later he again called; this time with the news that his fever had vanished, his appetite had returned, and that he felt a new man. Gradually, under large doses of iodide, the irregularity and enlargement of the liver disappeared, and he got quite well.

Some time ago I saw in consultation a young gentleman who had been travelling in unhealthy countries, and who, soon after his return, had fallen sick with fever. He insisted to his medical attendant that it was a malarial fever contracted on his travels, and persisted in taking quinine. Not improving, and still believing that his fever was malarial, I was asked to see him. Beyond a quotidian fever there was no evidence of malaria about him; but we elicited a history of a sore on the penis some months before. Having been told by the doctor he saw then that the sore was a soft sore and of no importance, he had not thought it worth while mentioning this circumstance before. We changed the quinine for mercury, and in a very short time the fever vanished.

Septic absorption of pent-up pus is a well recognised cause of quotidian fever, not unfrequently attended with marked rigor and diaphoresis. Sometimes the most careful examination fails to determine the situation of the pus. Now, if suppuration of this description occurs in an individual who has resided in malarious countries the case is almost sure to be diagnosed malaria. I have seen many such cases. Some time ago I was asked to see a young gentleman who for a long series of weeks had been the subject

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of a quotidian ague-like fever. He had travelled abroad. He had been seen by a number of eminent medical men, each of whom had given a different opinion, and prescribed an equal variety of treatment. I was asked to see the patient with the idea that the case might be malarial. I had no difficulty in pronouncing definitely against such a diagnosis, but what the nature of the case might be I did not pretend to know. Months later I met in the street a medical friend who devotes himself to throat and nose diseases. In the course of conversation he said, "By the way, you saw some time ago a Mr. So-and-so with Dr. ——?" "Yes," I said, "what about him? How is he? Is he dead yet?" "Far from that," my friend replied, "he is quite well. Some time after you saw him I was asked to examine his nose, which was bothering him, and by way of cleaning the passages preliminary to examination I syringed his nostrils with a warm alkaline solution. I did nothing more, and could see nothing wrong. But since that time he has had no recurrence of his ague-like attacks, and he is now quite well."

Some years ago I was asked to see a retired member of the Indian medical service, said to be suffering from malarial fever. I was told he had been treating himself, but had become alarmingly ill. On arriving at the house I found the patient insensible,—moribund, in fact. Another medical man had been called in by the wife, but neither he nor I for a time could get any light on the case. The wife could only tell us he had suffered much from fever both in India and since his return to England. We could find no evidence of enlargement of the spleen or other indication of visceral disease, such as might explain the situation. In looking round the room for bottles or other evidence of poisoning we came across a very ancient gum-elastic catheter. This gave the hint. On examining the patient's perinæum we discovered a huge abscess. This was freely incised—too late, unfortunately, to save the patient.

Pyelitis, catheter fever, pylephlebitis, empyema, phthisis, middle ear disease, elephantoid fever, the fever of early leprosy, the fever sometimes associated with gall-stones, these and many similar conditions occurring in patients from abroad, because they are accompanied by an ague-like quotidian fever, and because they occur in individuals who have been abroad, are constantly being diagnosed malaria, and treated as such. Therefore, I say, in approaching the diagnosis of a pyrexia in a possible malarial, while regarding tertian

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and quartan periodicity as absolutely conclusive, attach no weight whatever to quotidian periodicity.

(2) *The Therapeutic Diagnosis.*—Whatever may be the case in certain malignant infections in tropical countries, quinine, properly given, so far as my experience goes, never fails to arrest a malarial attack occurring in this country, and this within 48, or at most 96, hours of its administration. Therefore, a periodic or remitting fever that persists after adequate dosing with quinine, say, for four days, is not malarial.

Everyone believes, or says he believes, in the anti-malarial virtues of quinine, but it is strange how little confidence some practitioners have in its diagnostic value. Thus I frequently see in consultation cases of fever, presumed to be malarial, which have been taking quinine in large doses for weeks, or even months, and yet no suspicion about the correctness of diagnosis had been entertained. When asked to see a case of supposed malarial fever of a persistent nature, my first question usually is, "Has the patient taken quinine, for how long, and in what doses?" If I am told, as is often the case, that he has had large doses for many days, I feel sure, without seeing the case, that it is not one of malaria. In this country, if any of you are called on to treat a fever you suppose to be malarial, and if this fever resist quinine properly given and in full doses for three or four days, my advice to you is to revise your diagnosis; the fever is not malarial. I could quote many cases in illustration. It is hardly necessary to do so. The case of liver abscess I have already related is a case in point.

There are two important matters, however, which require to be attended to in applying the quinine test:—First, the dose must be adequate; second, the quinine must be absorbed.

In many instances quite an insignificant dose will check an ague. Many years ago, while in practice in the island of Formosa, I ran short of quinine, and had to economise my very limited stock. With this object I experimented to ascertain the minimum dose that would check an ague, and ultimately came down to half a grain three times a day. My Chinese patients, in several instances, said they were cured by this insignificant amount—so potent is quinine in some cases of malaria. This winter I had in hospital a patient suffering from double benign tertian infection. By a properly timed small dose of quinine it is sometimes possible to eliminate, in such a case, one group of the parasites without injuring the other

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group, and so to convert a quotidian ague into a tertian. I wished to demonstrate this to the students at the School of Tropical Medicine. I gave the patient three grains of quinine an hour, or thereabouts, before an expected paroxysm. Small though the dose was it killed out not only the group of parasites I proposed to eliminate, but the other group as well, and, contrary to my intention, cured for the time being the patient.

Quinine, as against the malaria parasite, is a very potent drug, but for reliable diagnostic, as well as for therapeutic, purposes it must be administered in much larger doses than those I have just mentioned. I would say that if a fever in this country resists 10 grains of quinine, given three times a day for four days, it is not malarial.

The dose of quinine should not only be adequate, but we must assure ourselves that it is absorbed. I am quite certain that many of the failures to control malaria by quinine are attributable to neglect of this essential. A catarrhal state of the gastric mucous membrane is usual in severe malarial attacks; such a condition is very unfavourable to absorption. Persistent vomiting is also common enough; food and drugs are alike rejected. To get the therapeutic action and diagnostic value of quinine in such circumstances the drug must be given hypodermically, or by enema.

Quinine nowadays is often given in tablet, or coated pill form; a most dangerous method in serious cases, and a most unsatisfactory one for diagnostic purposes. For some time quinine tablets have not possessed the flint-like hardness that used to characterise them a few years ago; they are now more soluble, but, even lately, twice I have seen them fail to be absorbed. In one case the patient developed a severe malarial attack on his way home from the Gold Coast. He took quinine tablets, and persisted in their use after his arrival in London. So much quinine did he take in this way without benefit, that both the patient and his medical attendant lost confidence in the drug, and the patient came very near dying. Three or four intramuscular injections of the acid hydrochloride broke the fever, and the patient quickly recovered.

In giving quinine, therefore, whether for therapeutic or for diagnostic purposes, we should assure ourselves that it is absorbed.

(3) *Microscopical Diagnosis.*—Of the three means for diagnosing malaria this is the most satisfactory, provided it is undertaken by one with large experience. A negative result by the amateur is

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valueless; a positive result is not much more to be trusted. It takes a large amount of practice to be able to recognise all forms of the malaria parasite, or to discriminate between them and vacuoles in the red corpuscles in fresh films, or dirt in dried and stained films. Articles in the medical papers by amateurs in malarial blood examination sometimes supply amusing reading. The frequency with which such writers encounter malarial rosettes and flagellated bodies, and free flagella, forms of the parasite least frequently met with, is very suggestive. Free pigment—in reality dirt—they often see. White blood-corpuscles I have known described as malarial parasites; blood platelets, as spores or sporulating parasites; cotton fibres or fibrin, as flagella. No one without some knowledge of the histology of normal blood, and much experience of microscopical examination of malarial blood, should attempt to write on the subject of the malarial parasite, much less attempt responsible diagnosis of malaria with the microscope. The thing is easy enough when one has been properly taught, and after large experience. Then the microscope is invaluable in diagnosis; but those who have not been so taught, and who have not had much experience, are eminently unreliable—they are often worse than useless. Therefore, for the majority of medical men, if they wish to have blood examined for the purpose of diagnosis in regard to the presence of a possible malarial infection, the services of an expert are necessary. But, although he may not be able himself to diagnose malaria with the microscope, every medical man should know how to prepare blood films suitable for such an examination. Very singular notions are entertained about this. I have sometimes had blood sent me in capillary tubes, such as are used to hold vaccine. I have also had sent me blood so thickly smeared on a slide that the preparation would not transmit light. I have also had sent me, from a distance, an ordinary slip and cover-glass wet preparation, with a request to examine it for malaria. Blood transmitted in any of these ways is useless. Unless the fresh blood can be examined at the bedside, or within an hour or thereabouts of preparation, dried films should be made, and subsequently transmitted to the laboratory for fixing, staining, and microscopical examination.

There are many ways of preparing malarial blood films. I have tried most of them. The best, I believe, is that described by Drs. Christophers and Stephens, or some modification of this. An

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ordinary microscope slip is carefully cleaned with alcohol. The patient's finger tip or the lobe of his ear is similarly cleansed. Finger or ear is then pricked with a clean and rather long ordinary sewing needle. A drop of blood is pressed out, and, as it may contain epithelial *débris*, wiped away. A second but very minute drop, the size of a large pin-head, is then expressed, and touched lightly (the skin must not be touched) with the clean surface of the slip about an inch from one end. Immediately the shaft of the needle is laid transversly on the droplet of blood so transferred to the slip. After waiting a second or two for the blood to run out by capillarity between the surface of the slip and the needle, the latter, held between finger and thumb, is drawn along the surface of the glass. In this way, with a very little practice, a beautifully fine and even film is produced. Half a dozen such films should be prepared. The single prick suffices, as the films can be made very rapidly one after another. After they have dried they should either be wrapped in very clean tissue-paper or placed in a dust-free slide-box, and transmitted to the laboratory. Here they are fixed with alcohol (five or ten minutes), and subsequently stained and examined.

For purposes of diagnosis I use either borax-methylene blue or dilute carbol thionine. These are very reliable stains, keep well, act rapidly, and give sufficiently good results. Undoubtedly one of the several modifications of Romanowsky's stain, when successful, gives a more brilliant picture; but they are somewhat unreliable, and unless the particular sample of stain employed is in frequent use, and known to be good, this method is not advisable for ordinary purposes of diagnosis. Leishman's modification of the Romanowsky method gives very beautiful results, and has the advantage of not requiring a preliminary fixation of the blood with alcohol, but it, too, has the objection of unreliability in ordinary clinical conditions. Malarial blood should not be fixed by heat.

In stained films, or in fresh blood, it is practically always possible to find the parasite—at all events in those cases of malarial infection that come to England. But this only on one condition, and this condition I would impress on you—the patient must not have taken quinine recently. You may find certain forms of the parasite —the crescent, for example—after full dosing with quinine, but the usual fever-causing forms, as a rule, rapidly disappear after

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administration of the drug. Therefore if you wish to have a reliable microscopic diagnosis in supposed malarial fevers, do not give quinine, or suspend its use for some time, before preparing the blood slides.

It is difficult to over-state the value of the microscope in the diagnosis of malaria. I could quote many cases in illustration. I shall mention two only: one showing what may be gained by employing it; one showing what may be lost by not employing it.

Some months ago I was asked to see a patient in the Great Northern Hotel. I was told he had just returned from abroad and was very ill. He had been sent by Dr. A. Scott, of Brighton, who, suspecting malaria, advised, as the patient was passing through London *en route* for his home in the country, that I should be consulted. I found an emaciated, delicate-looking lad, with an appearance suggestive of phthisis. I tried to get from him his history and symptoms, but he would not converse. On being asked a question he would smile in a vacant sort of way, and perhaps, after a long pause, emit a monosyllabic answer, but it was impossible to extract any useful information from him. Manifestly he was off his head. His parents informed me that he was 20 years of age, and had been in a cattle and fruit ranche in Shaster County, California, since the spring of 1900. He got on well on the ranche, liked the life, and kept his health till July, 1901, when, judging from his letters home, he appears to have had a malarial attack. For some time after this the parents received no news of their son. Becoming anxious, they telegraphed on October 9th for information, and received for reply: "Son has changed for the worse, but doctor gives hope." Immediately on receipt of this alarming telegram they set out for California, and, on arriving at the ranche, found that their son had been sent on October 5th to the State Lunatic Asylum at Napa. Here they found him on October 30th in a deplorably dejected condition, emaciated, and so weak that they feared he would not stand the journey home. His disease was stated to be melancholia and mania. On the way home he was better and worse. Twice or three times on board ship he had feverish attacks, which came on about 10 p.m., and lasted till 2 a.m., and for which the ship's doctor gave him some small white tablets with an "A" stamped on them, doubtless antipyrin or antikamnia. This was the story I got on November 19th from his parents. On examination I found the patient free from fever; his

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lungs were healthy, but his spleen was enlarged and slightly tender. I made films of his blood, which I examined the same afternoon. They showed many malarial crescents, and an abundance of the fever forms of the malignant tertian parasite. The patient was removed to St. George's Hospital and placed under Dr. Ewart. The same evening he had some fever, but full doses of quinine rapidly cleared his blood of the fever forms of the parasite. It was some weeks, however, before the crescents entirely disappeared. Under quinine the mental and physical condition gradually improved. There was no more fever, and he was discharged a month later free from malarial symptoms, but still somewhat slow in answering questions, and still mentally debilitated. I have little doubt that had the microscope been used earlier the true nature of this painful case would have been ascertained and suitable treatment instituted. Though employed dangerously late, I believe the microscope saved this lad's life, and perhaps his reason.

An officer of a liner trading to the East was brought on a stretcher to the Seamen's Hospital, Albert Docks, on July 30th, 1901. The history received with the patient was that his ship had just returned from the Mediterranean, where it had been running between Brindisi and Port Said, and that he himself had been in perfect health till three days before admission. He had never suffered from malaria. About that time he was walking about; but, looking ill, he was sent to his bed by the ship's surgeon. Presently he vomited blood, and passed large quantities of blood by stool, and, continuing to do so, was taken to hospital. On admission he was collapsed, with a temperature of 102·8°. Towards evening the temperature fell to 98·4°; hæmatemesis and melæna persisted. He had much abdominal pain, temperature fell to 96·4, the pulse became rapid, and he died next day. At the *post-mortem* examination the spleen was found slightly enlarged; the pancreas intensely congested and containing minute extravasations of blood; the mucous membrane of the stomach and upper part of the small intestine was similarly congested and studded over with ecchymoses. At the time the case was supposed to be one of hæmorrhagic pancreatitis. Pieces of the pancreas and intestine were preserved for microscopical examination. Owing to press of tutorial work at the School of Tropical Medicine, Dr. Daniels was unable to examine or make microscopical sections of the organs.

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But when, at the conclusion of the session, he had leisure to do so, he was astonished to find that the capillaries of the pancreas and intestinal mucosa were packed with sporulating malignant tertian parasites. Drs. Ross and Daniels have recorded and illustrated this very interesting case in the 'Journal of Tropical Medicine,' February 15th, 1902. None of us suspected malaria. The patient had not resided in any malarial place. There can be little doubt, however, that while his ship lay alongside the wharf at Brindisi, or, perhaps, during a short run on shore, this gentleman was bitten by infected anopheles, and thus received his fatal illness. Had his blood been examined earlier in the disease, or even within an hour or thereby of his admission into hospital, the true nature of the case could have been recognised, and the patient's life, perhaps, saved by timely hypodermic injections of quinine.

In rare instances, and at certain times in a considerable proportion of cases, the parasite, or its product melanin, even in severe infections, may not be detectable in the peripheral blood, and, as I have mentioned, even small doses of quinine, though therapeutically insufficient, may cause its disappearance temporarily, or, in the case of cure, for good. Even in these circumstances it would seem that the microscope may be relied on for a diagnosis.

It has been known for some years that during, and for some time after, malarial attacks the leucocytes are greatly diminished in proportion to the red blood-corpuscles. The diminution, however, is principally in the polymorphonuclears and lymphocytes, so that there is a relative increase in the large mononuclears. Drs. Christophers and Stephens, fixing their attention on this point, have marked out a method by which it is possible to give a fairly reliable diagnosis of actual or recent malarial infection, and this no matter whether quinine has been administered or not. The increase in the mononuclears, they state, is principally during the apyretic intervals, setting in as soon as the temperature begins to fall. They say, in a report to the Royal Society, that "an increase beyond 15 per cent. is proof of an actual or recent malarial infection."

Captain Leonard Rogers, I.M.S., after a very careful investigation of the point, endorses ('Brit. Med. Journ.,' April 5th, 1902) this opinion. He concludes: "An increase in the large mononuclears to about 12 per cent. and upwards, especially during the remissions of the temperature, strongly indicates malaria."

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Dr. Daniels, Superintendent of the London School of Tropical Medicine, has paid a great deal of attention to this point. In reply to a request for a statement of his views, he writes :—

"*Leucocytic Variation after Malaria.*—In all forms of malaria after, and to a smaller extent during, the fever, the large mononuclear leucocytes are relatively increased to the other leucocytes.

"This increase may be slight, and in certain cases has been so slight that they only formed 15 per cent. of the leucocytes. Commonly after malaria they vary from 25 to 30 per cent., and occasionally are over 40 per cent.

"The increase does not seem to be dependent on the species of parasite, on the number of parasites present during the acute attack, or on the severity of the symptoms or sequelæ. It is maintained for a period varying in different cases from two or three weeks to at least as many months, and slowly diminishes.

"It is not affected by quinine, either in large or frequently repeated doses.

"With some exceptions it affords a satisfactory proof of the probable recent presence of parasites.

"In normal blood it is rare to find more than 12 per cent., though I have seen as high a percentage as 16. In typhoid and Malta fever I have seen a similar increase, but it is exceptional. It is said to occur in small-pox. In certain diseases, particularly scurvy, there is also a marked increase; but in this disease there is also leucocytosis, and a more marked increase in the lymphocytes.

"The absence of this variation is of more value as a diagnostic point than its presence. As far as I have worked with the method, the only exceptions I have seen were when an intercurrent disease, such as pneumonia, which is associated with increase in the polymorphonuclear elements, also occurs. In other cases it has shown itself to be a reliable method for the exclusion of recent malaria.

"Working for three sessions with classes of students of 20 odd, many of whom, coming from the tropics, have suffered from malaria, this test has readily discriminated those of the students who have recently had malaria from those who have not.

"It has proved of value in differentiating cases of yellow fever from malaria (Low and Gray in St. Lucia).

"It has proved of value in differentiating hyperpyrexial fever of West Africa from malaria (Sims, Congo).

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"Though not of the same value as the presence of parasites, or of pigmented leucocytes, or recent pigment in the organs, it is of much higher value as a test of recent malaria than splenic tenderness or enlargement, and is of particular importance in cases where, either in the natural course of events, or after the administration of quinine, there are no characteristic symptoms.

"It is probably of about the same value as the agglutination test in typhoid, with moderate dilutions of the serum—say, 1 in 20.

"The main objection to the method is the difficulty that is sometimes present in deciding which are large mononuclears and which are lymphocytes. After actual practice this difficulty is small, and a column for undecided or doubtful leucocytes will be found useful at first. After a short time this will be, in the majority of cases, little used. In these determinations, however, there is a certain personal equation, so that counts made by different individuals vary within small limits."

These are Dr. Daniels' opinions, and, being based on large experience, I attach much value to them.

A few weeks ago I saw in consultation a gentleman recently returned from abroad. On the previous Saturday he was suddenly seized with a rigor and fever; on the Sunday he had no fever; on the Monday he had another paroxysm, and his temperature kept a degree or two above normal during Tuesday. I saw him on Wednesday morning. His temperature was then normal. He had received a few small doses of quinine. The tertian periodicity of the fever and a slightly enlarged spleen indicated a malarial attack, but to make certain I examined a film of his blood. As might have been expected from the quinine he had taken, there were no parasites, but a count of the leucocytes gave 20 per cent. large mononuclears, thereby confirming the clinical diagnosis. It is under such circumstances that a study of the leucocytic counts and variation will be of service in helping to correct diagnosis, and also in discriminating malaria from such conditions, as abscess of the liver, pneumonia and so forth, in which there is a leucocytosis; from typhoid, in which the lymphocytes are increased; and from other diseases with similar characteristic blood changes.

It is difficult to overrate the practical value of blood examinations. Only last week I had a striking illustration of this. A lady from India consulted me about what she described as an enlarged

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spleen. She had lived in a malarious district, and in years gone by had suffered a great deal from malarial fever; less so of late years. Medical men in India had told her she was "saturated with malaria." She came home on account of her health, especially on account of the enlarged spleen. She was rather emaciated, anæmic, and sallow. On examining the abdomen I found the pelvis and right side of the abdomen filled with a large, somewhat movable, rounded tumour. There was a notch low down on the right margin of the tumour, but there was an equally pronounced notch, if not two notches, on the left border. The tumour was not connected with the liver, and the whole of the *left* hypochondrium was resonant and soft. No indication of a spleen could be detected in the usual situation of that organ. The patient was positive, however, that the tumour began in the left hypochondrium, and that it was only recently that this part had become soft. Feeling puzzled about the case, I sent the patient to Mr. Meredith for examination of the pelvic organs, and, if thought desirable, for exploration or operation. Mr. Meredith was equally puzzled. The uterus and ovaries were not involved. We advised the patient to go to a nursing home with a view to exploration. At our first interview I had made some films of her finger blood, but being busy did not overtake their examination till two days later. You may judge of my astonishment, and of my thankfulness that our operative suggestions had not gone further, when, instead of finding a slight increase of large mononuclear white blood-corpuscles, indicating recent malarial infection, I found that our patient was suffering from advanced spleno-medullary leucocythæmia. The tumour we were puzzled about was undoubtedly an enlarged leucocythæmic spleen, which had, in consequence of its weight and size, become displaced.

We sometimes get assistance in concluding for or against malaria from considerations of time and locality. It is very commonly believed that a malarial infection may last a lifetime. Thus the old Indian, many years after he has come home for good, may tell us that he has recurrences now and again of a jungle (that is, malarial) fever contracted in youth. I do not believe in the malarial nature of such attacks. I have never been able to find the parasite in any such case. My belief is that malarial infection does not persist after three years from the date of invasion. Of course a patient can become re-infected—that is another affair; but I do not think

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the duration of any given infection exceeds three years. I will not be positive about this; we want more observations. But in my experience a malignant tertian tends to die out in from 18 months to two years, a benign tertian in about two to two and a-half years, a quartan in about three years. We know that other protozoal organisms having asexual and sexual phases require resuscitation by passing through the sexual phase every now and again; otherwise they become extinct. I believe the same to be the case with the malarial parasite. Other people besides the old Indian get feverish colds and different kinds of short febrile seizure, but because they have never been in India or Africa they call them influenza or what not. Had the old Indian never been to India he would do the same. He is rather proud of his jungle fever, and sticks to it longer than it sticks to him. While alive to the possibility of my being wrong in my belief, I am inclined to regard any fever which occurs three or more years after the patient has been away from the possibility of fresh malarial infection as non-malarial.

If a patient has never been to a malarial country he cannot get malaria, unless by artificial or abnormal means. In the malarial belt there are many non-malarial places—Malta, for example. A chronic or recurring fever from Malta in a person who has not visited a malarial place is probably Malta fever or enteric, certainly not malaria. Similarly, Barbados is not malarial. Therefore a chronic or recurring fever in a patient from Barbados who has never visited a malarial place cannot be malarial; most probably it is the fever symptomatic of some of the lesions in filariasis so common in Barbados. Commonly enough, because such fevers are ushered in by severe rigor, these filarial fevers are locally known as "ague." This is a misnomer, they have nothing to do with malaria.

I have seen a good many reputedly malarial patients from the Fen districts and other formerly malarious spots in England. On critical examination not one of them has turned out to be malarial. They were assumed to be malarial because there was some periodicity about the fever, perhaps enlarged spleen, and because they came from localities which were formerly malarious. I have never seen a case of indigenous English malaria. I believe the plasmodium has died out in England, like the wolf and the wild boar.

In concluding these rambling and disconnected, but I trust practical remarks, I would again impress on you the importance

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of a thorough reliance on the three classical methods of diagnosing malaria:—The clinical—tertian and quartan periodicity; the therapeutic—the curative effect of quinine; and the microscopical—the detection of the parasite or its products in the blood, and the characteristic leucocytic variation. A thorough and intelligent application of one or all of these methods seldom, if ever, fails to lead to a correct diagnosis for or against malaria. At the same time I would warn you that although a positive diagnosis of malaria may be indicated in some given case, the presence of malaria does not exclude other diseases. On the contrary, malaria often predisposes to other infections. Similarly, the explosion of a latent malaria is favoured by other infections, especially typhoid and tuberculosis.

The PRESIDENT extended to Dr. Manson the cordial thanks of the Society for the paper, and their high appreciation of the value of it. It had been the custom for some years past that the recipient of the Fothergillian gold medal given by the Society should, on the last meeting of the Session, deliver an address concerning the subject for which the award had been made. If any justification were needed of the action of the Society in making the award, that would have been fully supplied by the paper to which Fellows had just listened. It would be impossible to over-estimate the importance of such a paper, and the practical teaching which it embodied. The diseases which Dr. Manson had dealt with he had presented as a master, and with a knowledge with which no one else's would, perhaps, compare. Most of one's knowledge on the subject was founded upon what Dr. Manson himself had taught. The ordinary physician seldom had any real practical acquaintance with the diseases under consideration, and therefore the rules laid down were of the greatest help. One could not but feel thankful that, by observing those rules, one was able to extend some benefit to one's fellow-creatures, and thus in some measure remove the somewhat pessimistic reproach contained in Dr. Manson's opening remarks.

Dr. T. D. SAVILL said he could not pretend to have had much experience of malaria, but he remembered that Dr. Charles Murchison used to point out, as Dr. Manson had done now, that it was commoner to mistake cases which were not malarial fever than the converse. The last clinical lecture which Dr. Murchison gave was on that subject, and appeared in 'The Lancet' in April or May of 1879. Murchison there pointed out that there were 13 different causes of quotidian intermittent pyrexia, any of which might be taken for true malaria. One very important lesson of the present paper was that every practitioner should be able to easily prepare blood films for examination by experts. This could be done readily at the bedside. There was a very easy method of making really good films, which he learned in Vienna, viz., to take an extra thin oblong cover-glass, put a spot of the blood on one half of the oblong, holding the glass by the edge, lay another glass on the top (half covered by half) till the blood spreads out, then draw them apart suddenly. He had never found it distort the corpuscles as some alleged. By the use of Leishman's

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stain and distilled water one could prepare one's own stained specimen, and with a little practice could get a doubly-stained specimen, showing the polynuclear and mononuclear cells, and all the other information required.

Dr. SAINSBURY asked what Dr. Manson regarded as an adequate dose of quinine when given by the hypodermic method.

Dr. BEEVOR said that a few months ago, at the Great Northern Hospital, he had a man who had been in South Africa, and who was admitted with attacks of shivering. The man said it was malaria, as he had had that disease before, and he was quite familiar with the symptoms. As he had the attacks every day, and as they did not give way to large doses of quinine, he (Dr. Beevor) had the suspicion that the case might not be one of malaria. On further examination the man was found to have a perineal abscess. That was opened, and the shivering fits then entirely disappeared. That illustrated what Dr. Manson had mentioned as to taking a patient's own diagnosis.

Dr. MANSON, in replying on the discussion, said he had given up the practice of making blood films on cover-glasses for several reasons. Cover-glasses were very fragile and very difficult to clean, and when dealing with large numbers of specimens one got impatient at the number of breakages. He now always used slips, which could be employed very simply, and it was possible to attach a label to a slip. If specimens got mixed up, the result might be very serious. Everyone knew the difficulties caused by mixing up bottles of urine in one's consulting room, and the same could happen with blood films. He had referred in his paper to a case of splenic leucocythæmia, the nature of which he discovered before surgery was practised; this, as it happened, was very nearly an instance of serious confusion resulting from names being almost obliterated from specimens of blood. He agreed that Leishman's stain was a good one if it were constantly in use and was known to be fresh; but if it were required only once a week or less, it was very unreliable, and an unreliable stain was worse than no stain at all. Thionine and borax methylene blue he found never went wrong, and though he recognised the value of Leishman's stain for demonstrating the minute structure of the malaria parasite, he did not rely on it for purposes of diagnosis.

With regard to the dose of quinine for hypodermic injection, he looked upon 7 to 9 grains as adequate. Very few cases of malaria resisted one or two such doses.

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May 26th, 1902.

THE ANNUAL ORATION—"THE POWERS OF NATURAL RESISTANCE, OR THE PERSONAL FACTOR IN DISEASE OF MICROBIC ORIGIN."

By STEPHEN MACKENZIE, M.D. Aberd., F.R.C.P. Lond.

MR. PRESIDENT AND GENTLEMEN,—There can be few amongst us who do not rejoice in the privilege we have of living in an epoch of such progress and mental activity as the present. The great increase of knowledge of the latter half of the last century is found in every branch of science, but it impresses us, perhaps, most in the advance of scientific knowledge in our own profession. It has been brought about by improved means of exact and minute research aided by instruments of increased precision. So that if medicine, in its widest sense, cannot be claimed as an exact science, our methods of dealing with the complex and difficult problems of disease in the living and ever-changing body are as exact as the present state of science permits. Great as have been our advances, we cannot doubt that they will be infinitely greater, and probably more rapid, in the future. Many, however, are dazzled and led away by the apparent completeness of our recently acquired and almost daily increasing knowledge, and are inclined to overlook, to neglect, and almost to deride, the knowledge which we have acquired by the patient industry and accumulated experience of our predecessors—the work that our fathers have given the best years of their life to accomplish. Our President (Dr. W. H. Allchin) touched upon this point in his address last year. There could be no greater or more inexcusable mistake. Whilst gratefully acknowledging and utilising the newest methods and knowledge as aids to medicine and surgery, theoretical and practical, we must consider them in relation to the observed facts, empirical and scientific, that have been acquired since medicine has been studied as an art or a science. Some of the aphorisms of Hippocrates, the clinical observations of Sydenham, and the pathological observations of John Hunter, are as pregnant of truth and as worthy of being treasured in the memory now as when they were first promulgated, and are in no sense less important than are the most recent discoveries in the

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laboratories of the bacteriologist and the chemist. It is quite certain that the accurate record of careful observations of the phenomena of disease will always be valuable additions to medical knowledge, whether or not capable of explanation or use at the time they were made. We see striking illustrations of this in the writings of the three great Guy's physicians—Bright, Addison, and Hodgkin. There have been many stages in arriving at the knowledge which we at present possess. The progress of morbid anatomy and the revelations of the bacteriologist and chemist have given great precision to our knowledge, and have opened out new methods of research and treatment. The task which I have set myself on the present occasion is to point out that whilst availing ourselves of all scientific discoveries, we must not overlook or forget the valuable knowledge which we already possess, and may still further acquire by careful, patient, and persevering clinical work. The tendency of the medical mind at the present time seems to be to attach too great importance to the germ or seed, and too little to that of the body or soil, in diseases of microbic origin. I propose to pass under review a few diseases of this class, and to endeavour to point out the share or relative importance of these two factors in such diseases.

Influenza.

Let us take the case of influenza. It presents the extreme end of a series of infectious diseases—*i.e.*, diseases of microbic origin. It prevails pandemically at irregular and often long intervals, usually followed by epidemics for some years. When pandemic, it is remarkable for the number of persons who are attacked, and the rapidity with which it spreads from country to country. Neither race nor class, sex nor age, affords exemption. It is so widely spread when pandemic that very few escape, though probably a very few are immune, and these attribute their escape to some nostrum or the disinfectant most in fashion. At these times the seed is everywhere, and almost everywhere it finds a congenial soil, so that the natural resistance or personal factor, as far as susceptibility goes, is practically nil. Not so the effect; though said to be the smallest bacillus known, like the Biblical grain of mustard-seed it gives rise to very great results. In the aged, in those suffering from pulmonary and cardiac disease, and in those devitalised from any cause, it is a very serious malady, often

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leading to a fatal issue. Though in the robust it is often apparently a comparatively trivial disease, this generation has learned that it is to be treated with great respect, and that apparently slight cases neglected may lead to very grave results. We have learnt, too, that it may be attended with very serious and dangerous complications, such as abscess of the brain, purulent otitis, phlebitis, affections of the heart and nervous system, &c. Whether these complications are the direct effect of Pfeiffer's bacillus, or are due to a mixed infection, has not been fully worked out.

The subject of mixed infections raises a very interesting question of great importance in several of the infectious diseases, indicating that the seed is not always pure, or that the tissues, the resistance of which has been broken down by one microbe, fall a ready prey to others; but into this point I do not propose to enter.

Lastly, we have learnt that the effects of an attack may be of prolonged duration, continuing even for years. Whether they depend on tissue changes effected by the direct action of the bacillus or its toxins in the attack, or the latter surviving long after the attack of influenza, is uncertain; but we see in diphtheria the supervention of paralysis some weeks after the acute stage has passed off, and in syphilis the occurrence of nervous lesions years after all other active manifestations which we recognise as directly syphilitic have ceased to present themselves. Let us turn now to another subject—tuberculosis.

Tuberculosis.

When I began the study of medicine we were still in the throes of battle as to what was tubercle and what tubercle was. The unitarian doctrine of Laennec did not by any means meet with universal assent. The microscope was pressed into service to decide what was the essential constitution of tubercle, and diligent search was made for the "tubercle cell," as there had been for the "cancer cell," as fruitless in the one case as in the other. Then followed the epoch in which Villemin showed that tubercle was inoculable, though many pitfalls were encountered in establishing this fact. Finally, in 1882, Koch was able to demonstrate that the specific cause of tuberculosis in all its various forms was a micro-organism, the bacillus tuberculosis, that it was present in every tuberculous lesion, and that without this there was not any

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tuberculosis. The gropings to establish its nature, often highly scientific, sometimes blind, were at an end; the efforts of many of the keenest and ablest workers to understand its morbid anatomy and pathology were harmonised and their errors were corrected, and we obtained an absolute criterion to which we could appeal to decide what was tuberculous. It was left to the clinician and sanitarian to apply this knowledge in the treatment and prevention of disease. With the aid of the bacteriologist we have learned a great deal of the life of the tubercle bacillus, both inside and outside the body, and of the ways by which tuberculous persons become affected. But in this respect the tendency has been, perhaps, to concentrate attention on the microbe, and to devote less consideration than its importance demands to the human host in which it finds a lodgment. This is seen not so much in the deadhouse, where the gross changes in the organs are forced on our notice, as in the wider aspects of the subject which deal with the causation and prevention of tuberculosis. We are apt to think too much of the *seed* and too little of the *soil*. We have learned, since Koch's discovery of the bacillus, that from many sufferers from tuberculosis, more especially in pulmonary phthisis, the secretions or discharges given forth contain the bacillus in an active condition, and that the clothes and handkerchiefs and the beds and the floors and walls of the dwellings of the sick are contaminated in this manner so as to be a source of danger to others. These secretions and discharges undergo desiccation, and are commingled with other particles, constituting dust, which are inhaled with the air we respire, and haply finding in the respiratory or digestive organs conditions favourable for their growth, increase, and multiply, and bring about tuberculosis. We have reasons for believing that casual bacilli may not only reach the respiratory and digestive organs, but may make their way by the blood-vessels into practically all parts of the body, where, in favouring conditions, they set up tuberculous processes. Thus are accounted for cases of tuberculous meningitis, tubercle of the kidney, &c. We have learned that the bacillus tuberculosis may be contained in the milk, the butter, the cheese, and the flesh which we consume as food. In recent years great efforts have been made to exclude this mode of infection by the extermination of tuberculosis in cattle and the destruction of foodstuffs derived from infected bovines. Like a bolt from the blue came the recent pronouncement of Koch himself at

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the late British Congress on Tuberculosis, that the risk of transference of tuberculosis from cattle to human beings was so slight as to be a negligible quantity. The gauntlet thus thrown down was immediately taken up. A Royal Commission has been appointed to consider and to report on the subject. Already evidence has been tendered that such transference does take place—a fact not disputed by Koch himself. Many must admit that the evidence not only as to the fact, but also of the frequency with which tuberculosis is derived from bovines, is in need of revision and consideration. Dieudonné, Cattle, Shennan, Still, Carr, and Colman have shown that in the tuberculosis of infants the primary seat of infection in most cases is through the respiratory and not the digestive tract; and further, Koch, in his address to the Congress, said: "It is just as likely that these cases of intestinal tuberculosis were caused by the widely-propagated bacilli of human tuberculosis." Cattle writes: "It is not only milk that goes into the mouths of children; they grub about on all-fours, plastering their fingers with the tuberculous dust hidden away in inaccessible corners, and then put their fingers in their mouths." It is more than possible that the danger of infection from food has been over-estimated. It is none the less our duty to use all the measures which experience dictates to prevent the entrance of the bacilli by this route, and we should be guilty of folly if we relaxed the measures of prevention now in use until we have a decisive verdict on this point.

But it is of the *soil* in which the tuberculous seed grows that we have also to take account. We have learned that the bacillus tuberculosis is so widely disseminated that no one is exempt from the risk of infection. The dust inhaled in many dwellings, in public rooms, in vehicles, and in the streets is often contaminated with the bacillus. Strauss has shown that in cotton-wool plugs in the nostrils of those in attendance at the Charité Hospital, Paris, tubercle bacilli were found in nearly a third of the cases examined, and that they proved infective to animals. Dieudonné found tubercle bacilli and pyogenic cocci on the hands and in the noses of children from the age of nine months to two and a half years, whose fathers or mothers were the subjects of tuberculosis. Though the protective arrangements at the entrance to the respiratory passages to some extent prevent the entrance of the bacilli, this protection is by no means complete, and when adenoids or some

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malformation of the nostrils compel oral breathing it is very imperfect. This is shown by the great frequency of pulmonary tuberculosis. *Post-mortem* experiences reveal to us that tuberculous changes are found in the lungs and other parts of persons dying from various non-tuberculous diseases or killed by accident—placed by different writers at from 30 to 90 per cent.—but which have become arrested and obsolete. So that not only are all liable to become infected, but we know that in a much larger number of persons than was formerly imagined actual infection does take place. But we learn from this fact also that the infection may be but slight and temporary, and that within the body and in the tissues there exist physiological mechanisms that arrest the invasion and bring it to a standstill. We know also that where the risk of infection must be considerable, as in the aggregations of consumptives in hospitals, very few who move and live among them contract the disease. Though the experience of all hospitals has not been so favourable as has that of the Royal Hospital for Consumption at Brompton, it has been stated that over a considerable number of years the incidence of phthisis amongst the physicians, nurses, and servants of that institution has not been greater than that of the general population.

Thus it is demonstrated that the human soil is not in all persons a favourable one for the growth of the tuberculous seed, and that the human organism has a considerable power of resistance and self-protection, very happily illustrated by Osler by the 'Parable of the Sower.' Dr. W. Howship Dickinson has within the last month published an able and interesting sermon with St. Matthew's 'Parable of the Sower' for his text. Dr. Dickinson has treated the subject of tuberculosis on almost exactly the same lines as myself, but in his own peculiarly felicitous manner and literary style. It is equally obvious from the great frequency of tuberculous diseases that all are not equally fortunate in this respect—*i.e.*, in the powers of natural resistance; or, in other words, some are more vulnerable than others. Mankind is naturally resistant to tuberculosis (Kanthack). On what this vulnerability immediately depends we need not here discuss, but we know a great deal of the circumstances in which tuberculosis occurs. We know that it may occur at any age, that its various forms have special age proclivities, and that the majority of deaths from tuberculous affections, as a whole, occur between 20 and 30 years of age. We know by the writings

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of our predecessors and by our own experience that many inherit and some acquire a tendency to tuberculosis. There can be no doubt that the progeny of the tuberculous are much more liable to tuberculosis than are those in whose family no such strain runs. On what that predisposition depends opinions may differ, and we have no absolute knowledge. Whilst congenital tuberculosis, whether placental or germinative, is an established fact, it is extremely rare, but most who are born of tuberculous parents and become tuberculous are not, we believe, actually so at birth. Inheritance in bovine tuberculosis is believed to be answerable for more than one-half of its very considerable mortality. We are often able to detect the tendency to tuberculosis in individuals by their conformation, and we know that they often show a marked proclivity to catarrh. I do not discuss the nature and significance of what used to be called the strumous or scrofulous diathesis. We are also quite aware that pulmonary and other forms of tuberculosis do occur in those of robust physique with well-formed chests, but this does not detract from the importance of our experience that the delicate and the frail, or those of feeble vitality, are the most prone to become tuberculous. The great liability to catarrh in the delicate offspring of tuberculous parents by causing inflammation of the air-passages doubtless weakens their powers of resistance to the tuberculous bacillus which is nearly always at hand. Alcohol again is a predisponent. How often do we hear in taking the history of a family, otherwise clean as regards tuberculosis, the statement that a relative died from consumption, but that "he gave way to drink" —an explanation that in the minds of his family this was the cause of his becoming tuberculous? It has been shown that those who are intemperate or who have opportunities of drinking show a higher mortality from phthisis than do others, and that the children of the intemperate are more prone to become tuberculous than are those of the temperate. No doubt through alcohol persons acquire or inherit a lessened power of resistance to tuberculosis. Diseases like measles and whooping-cough, which are often complicated with broncho-pneumonia, and diabetes, which greatly reduces the powers of vital resistance, may open the door to tuberculosis.

In the exactness of our recently-acquired knowledge of the pathology there has been a tendency, as I have already stated, to deal lightly with the accumulated experience of the past, and to set

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little esteem upon the laborious work of the great masters of medicine. A timely protest against this tendency has been recently made by Sir Dyce Duckworth, in his admirable address on "The Personal Factor in Tuberculosis," which deals with this important subject with an ability and length that I cannot command, and which I have read since writing this oration. The personal factor in the clinical aspect and in the pathology of tuberculosis is of quite equal importance to that of the microbe, essential as that is for the production of tubercle. "The microbe," writes Professor Vaugeley, of Bordeaux, "is nothing whilst the organism in which it thrives is the main factor." How thoroughly this is borne out in the treatment of tuberculous diseases. When once the seed has taken a firm root in the body we cannot with certainty at the present time kill it. It may grow or it may wither, we may cut it out when it is purely local and accessible, in some parts we may destroy it by Finsen's light treatment or Roentgen rays when it is confined to the skin and superficial mucous membranes; but when deeply seated we at present know of no remedy which will certainly destroy the virus, not even lachnanthes. I say at present, because I do not think we ought to be hopeless of effective results from tuberculins or antitoxic serums. It is true that the hopes that were raised by Koch's first tuberculin, prematurely published and boomed by the lay press, did not realise the expectations that were excited. No doubt the pendulum swung round too completely, and the first trials insufficiently tested, or used only in the manner then advised, did not offer encouraging results. Nor has general experience, so far as it has been employed, been much more encouraging as regards Tuberculin R. Koch has introduced a still newer preparation, and there are some who hope that with modifications and in smaller doses distributed over a considerable period, especially in early cases of phthisis, it may yet prove effective. Petruschky claims to have obtained good results by much smaller doses than were originally employed, and extending over several courses with intermissions. The method is scientific, and is supported by our experience in rabies, tetanus, and diphtheria. Some remedies, such as creasote and guaiacol, of undoubted service in the treatment of phthisis, though not strictly bactericidal in doses that can be safely administered, may possibly diminish the activity of the bacilli and lessen or prevent secondary tuberculosis of the digestive tract by their action on the bacilli in swallowed sputa,

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but in the opinion of many they only do good by their action on mucous membranes and by lessening cough and expectoration. We disinfect or destroy the sputa and other discharges from the tuberculous, not for the good of the patient, but to prevent the dissemination of the disease to others. Practically our treatment of tuberculous diseases that are not accessible to the surgeon consists in increasing the vigour and resistance of the patient, by life in the open air, abundance of sunlight, which is of great importance, as its chemical rays are strongly bactericidal, thorough ventilation of the apartments of the sick, and by choosing a climate especially suitable for each case when practicable; abundant, very nourishing and oleaginous food, and by strict attention to general and personal hygiene. In fact, our principal object and power, whether we recognise it or not, is to endeavour by every means in our hands to raise the natural resistance of the body to the microbe—to devote our attention to rendering the soil an unfavourable one to the growth of the tubercle-bacillus. It is encouraging, and sometimes astonishing, how successful our efforts are in this direction, especially when the circumstances of the patient allow of the full use of the means experience has taught us. It is, however, I think, too great a stretch of the truth to say, as has been said, that "tuberculosis is one of the most curable of diseases." The wise do not often talk of "curing" tuberculosis. Tuberculosis may become arrested under our treatment, and often without it, but we can never be sure that a quiescent or obsolescent tuberculous process may not resume activity, like Mont Pelée, with eruptive violence under certain circumstances. We welcome and admire the princely gifts of Sir Ernest Cassell and other philanthropists for the establishment of sanatoriums for the treatment of the consumptive, and we recognise the duty of municipal and other bodies taking steps for carrying out the "open-air treatment" of phthisis so as to extend to the less affluent the benefits which otherwise are confined to the rich or well-to-do. Though the open-air treatment was first practised in England by Dr. Boddington, it did not meet with general adoption, and this country is at the present time behind others in the means for carrying it out. There is now, however, an awakening to the importance of this method of treatment.

A point, however, to which I most particularly desire to draw attention is that the undoubted and great diminution in the

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mortality from consumption that has been going on during the last 50 years, not only in this country, but also in other countries where statistical evidence is available, *preceded* the almost exact knowledge which we have now obtained of the pathology of tuberculosis and the discovery of the bacillus. The great value of the work of the late Sir George Buchanan should always be remembered in connection with his reports on the conditions under which consumption was most prevalent. This striking diminution in the mortality of consumption has been steadily going on under many unfavourable conditions, before any effective measures were taken for disinfecting or destroying the sputa or separating the sick from the healthy. During the greater part of this time in numerous cases husband and wife, mother and daughter, sister and sister were occupying the same bed when one has been the subject of consumption. It has gone on, too, in spite of the great migration from the country to the towns. There can be no doubt that this lessening of consumption has been due to improved sanitation, to better draining of the land, to improved disposal of sewage, and to the better housing, clothing, and feeding of the general population. Of course, we recognise the great influence of modern surgery which, dealing with tuberculosis of the bones and the joints, the glands and the serous cavities, has saved a great number of tuberculous subjects from becoming consumptive. The greatest triumphs in tuberculosis, as in other diseases, have been in *prevention.*

What lessons do we learn from this general survey of the share taken by the seed and the soil of tuberculosis? First, that we have to prevent or lessen the diffusion of the disease by preventing the dissemination of the tuberculous germs. This is to be done by segregation of the sick, either in institutions especially arranged for their care, or, when this is impossible, by providing that they live in their homes in well-ventilated apartments not occupied by the healthy. We have also to secure the destruction or disinfection of the sputa and other discharges containing the virus, and the subsequent disinfection of the rooms that tuberculous subjects have occupied. These measures we are all keenly alive to, and we have to continue and to increase our efforts to this end. But we have, in addition to taking all means in our power to prevent the propagation and dissemination of the seed, not to lose sight of the soil in which it breeds. This is the second direction in which efforts must be made if we are to succeed to lessen still further the prevalence

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and mortality of tuberculous diseases. What we have to do in this respect is obvious, but not easy. We have to increase the natural powers of resistance to the action of the specific microbe both in the individual and in the general population. We have to prevent overcrowding, and to improve the dwellings of the working classes and the poor. Poverty is a powerful predisponent to disease of most kinds, but especially to tuberculosis. Koch says: "The breeding-places of the tubercle bacillus are in the slums." Sir J. Crichton Browne has said: "Tuberculosis stands in inverse ratio to the rateable value of the property." I remember many years ago, before the discovery of the bacillus, someone pressing the late Dr. H. G. Sutton to define what tubercle *really* was. He replied "Tubercle is making shirts at 5*s*. a week." We cannot make the poor rich, but it is our duty, no less as citizens than as physicians, to see that the poor and the working classes are decently housed, and that their dwellings are kept in a proper sanitary state. We must bring the pressure of public opinion to bear upon municipalities, urban and rural councils, to improve the conditions under which the poor live. Rich and poor alike must be still further educated as to the imperative necessity of fresh air by means of the "open window" or more scientific methods of ventilation. We must secure not only for the dwelling, but for all places where people congregate—workshops, factories, churches, and theatres—that they do not respire re-breathed and contaminated air. We must use our influence as medical men to prevent consumptives from marrying and propagating offspring predisposed to the disease; we must continue to urge temperance; we must endeavour to rear a population of more robust physique, offering a more resistant soil to the seeds of tuberculosis. What we have to aim at is the improvement of the race. This is a very high, and to some it may seem a transcendental, ideal, involving great economic and social problems. But the material prosperity of the country has steadily increased. The poor are better housed, clothed, and fed than they were formerly, and we have the encouragement of the past in the last half century teaching that tuberculous diseases have materially diminished by the efforts that have been made in these directions.

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Rheumatic Fever.

I pass on to the consideration of rheumatic fever. Here our ground is less secure, as the evidence of its bacterial origin is not absolutely admitted at the present moment, though it appears to be within almost immediate realisation. Rheumatic fever is a common disease in this country. It has received a great deal of attention at the hands of medical observers, and has exercised the minds of most of the thinkers in our profession. The state of our knowledge 30 or 40 years ago may, I think, be fairly summarised by saying that it was believed and taught that rheumatic fever was an acute non-puruleut inflammation of many joints and of fibrous tissues, attended by fever and acid sweats, and liable to be complicated by endocarditis and pericarditis; that it was due to an acid in the blood, and was to be treated by alkalies. A nervous theory was put forward to explain some of the symptoms, but it never took firm root. Prout suggested that lactic acid was the cause of the disease, and B. W. Richardson endeavoured to prove this experimentally, but this evidence is not accepted. Latham, a few years ago, explained how lactic acid might be formed in the body. The acid theory was at one time so firmly established as to appear impregnable. It was, however, but a remnant of the humoral pathology that at one time dominated medicine. The faith of most was shaken when Gull and Sutton published their well-known paper on "The Treatment of Rheumatic Fever by Mint-water." They revealed to us, almost for the first time, the natural history of the disease untreated by drugs, and showed that it ran very much the same course, and lasted about the same time, as when treated by alkalies or other active medicines. This was, of course, before the introduction of salicin and salicyl compounds into the treatment of rheumatic fever. The kindly banter of Wilks, who said that "the use of alkalies in rheumatic fever was to remove the idea of acidity from the mind of the doctor," probably did not a little to help in the same direction. We have since learned, by the gradual growth of medical knowledge, to take a wider view of the nature of rheumatism. The original conceptions were founded on descriptions of the disease as seen in the adult, but the study of the disease in childhood has enlarged our grasp of its clinical features, and we know that, whilst acidity of the urine and the sweat is characteristic of rheumatic fever, it occurs also in other

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acute febrile diseases. But even if an acidity of blood, or rather a diminished alkalinity of the blood is true, it has still to be ascertained how this is brought about. It is interesting to note that Poynton and Paine found that the organism which they have found in rheumatic fever grows best in an acid medium. We have learned that rheumatism, like tuberculosis, is often an inherited or family disease, that it has many expressions besides the arthritis, and that many conditions, such as endo- and pericarditis, some cases of pleurisy, several affections of the skin, tonsillitis, and chorea may occur independently of arthritis, and have as much right to be regarded as rheumatic as the joint affection. I am aware that this position is not universally admitted, but though the difficulties of obtaining evidence on this point are great, it is those who have devoted most attention to the subject who, as a rule, are most convinced of its truth. But those who hold this view, equally with those who do not see the facts in the same light, have always recognised that there must be another factor besides predisposition to account for the occurrence of rheumatism. What this *tertium quid* is has exercised considerable thought and speculation. The influence of the prevailing pathological doctrine has naturally suggested the possibility of a microbe. This is no new view, but was maintained years ago by Hueber, Strümpell, and others. It is admitted or suggested that many or most of the clinical phenomena connected with this disease fit in with the microbic theory, and that the morbid anatomy is not opposed to it. Rheumatism is mainly a disease of the first half of life; age exercises a great influence on the form in which it manifests itself. The acute febrile and self-limited course of rheumatic fever corresponds to that observed in other fevers of ascertained microbic origin, whilst the non-febrile or only slightly febrile course of some of the conditions claimed to be rheumatic, such as chorea, is paralleled by diphtheritic paralysis and other nervous affections due to toxins. The occurrence of post scarlet fever rheumatism appears to be an example of "the door being opened" (to use the happy expression of Sir William Broadbent) by the former to the latter, as it is by influenza to typhoid fever. The occurrence of several cases of rheumatism in its various forms in succession in the same house, not confined to members of the same family, but in subsequent occupiers, recorded by Mantle and other observers, is strongly in favour of the infective—that is, microbic—nature of rheumatism. Newsholme's valuable work points in

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the same direction. He has noted the epidemic or semi-epidemic prevalence of rheumatism. He found that this is associated with lowness of subsoil water and relatively high temperature of the earth, conditions which probably influence either microbic growth or dissemination. Seasonal influence is considerable, as in typhoid fever and cholera, the greater number of cases of rheumatic fever in London occurring in October and November. Rheumatic fever, like typhoid, confers no immunity to subsequent attacks. On the contrary, a person who has once had rheumatism in any of its forms is extremely likely to have it again, but whether this is due to increased proclivity or simply indicates personal predisposition there is no evidence to show. Several observers, such as Mantle, Achelme, and others, have found micro-organisms in the inflamed joints and blood in rheumatic fever, but their results were not pushed to the point of absolute certainty. Wassermann, Westphal, and Malkoff abroad, and Poynton and Paine in this country, have found a diplococcus which agrees in its general characters. The two latter have obtained this diplococcus in pure culture from human rheumatic lesions, and have reproduced by intravenous inoculation in the rabbit endocarditis, pericarditis, carditis, pleurisy, pneumonia, nodules, and a nervous condition resembling chorea, as well as polyarthritis and inflammation of the sheaths of the tendons, and have also found the same organisms in the induced lesions.

An observation of great interest made by Poynton and Paine is that in a culture from a "sore throat" in a patient with muscular pains, a history of a previous attack of rheumatic fever, and valvular disease the diplococcus was found. It was isolated and cultivated, and was injected intravenously into a rabbit, producing in that animal mitral valvulitis and great dilatation of the heart. This observation, and the fact that sore throat so commonly occurs as an early symptom of rheumatic fever, suggest that the usual point of entrance of the virus is the tonsil or the pharynx. The observations of Poynton and Paine, especially the experimental evidence, appear so complete as to be convincing. We require further confirmation of these observations before the microbic theory can be said to be established beyond dispute, but Osler and Allchin have boldly transferred rheumatic fever to the category of acute infectious diseases. If confirmed, as I believe these researches will be, what a valuable aid they will be to the precise

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understanding of the pathology of rheumatism. When the seed is fully identified we may hope to learn from its life-history outside the human body means for the prevention and treatment of this disease.

But I wish to point out that if the bacterial nature of rheumatic fever is fully established, the patient work on the clinical side will not have been thrown away, that we shall still have to take account of the soil no less than of the seed, and, as in tuberculosis, the personal factor will remain as important in the future as it has been in the past. If the bacterial nature of rheumatism is conclusively proved, we shall still have to take into account individual liability to the disease, and to recognise that whilst some persons exposed to wet and cold, especially when combined with fatigue and exhaustion, escape, others similarly exposed are prone to contract rheumatism. Of course, it is well known that many cases occur without such exposure. But Pasteur, Wagner, and others have shown that by exposure to cold fowls (naturally immune) become susceptible to anthrax infection, and Monti has demonstrated that dogs, naturally more resistant than man to the pneumococcus, are taken ill and die from pneumococcic infection solely when they are inoculated after giving them a very cold bath. These facts are suggestive in connection with the microbic nature of rheumatism. We shall continue to find that the predisposition runs in families and is usually inherited. We shall, in fact, find that the soil is no less essential for the production of rheumatism than is the seed.

Appendicitis, or Perityphlitis.

Appendicitis, or perityphlitis, offers an interesting and instructive study, clinically and pathologically, in connection with the theme which we are considering. From being regarded, as it was when I commenced the study of medicine, as a rather rare condition, it has come to be regarded as a very common one, not only by the profession but by the public. Whilst the less acute or less severe forms have been recognised for a long time, we have in recent years learnt that the very acute and fulminating cases due to ulceration and sloughing of the appendix, causing abscess and perforation, account for a great many obscure cases of peritonitis, the nature of which was unsuspected and misunderstood. The increase of our knowledge on the subject has reached a high degree of precision through the observations of the surgeon, the pathologist,

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and the bacteriologist. It was, I believe, Sir Samuel Wilks who, in the first edition of his work on 'Morbid Anatomy and Pathology,' emphasised the fact that in inflammation and abscess in the right iliac region—perityphlitis—it was usually the vermiform appendix that was at fault, and not the cæcum as was then generally taught. To the surgeons in this country and in America we owe the great extension of our knowledge that in inflammatory affections of the abdomen the appendix was at fault with a frequency not previously imagined, and they have pointed out the anatomical conditions in the appendix that favour and predispose it to disease. The bacteriologists have determined the micro-organisms that play such an important *rôle* as the excitors of the inflammation and its consequences in the appendix. Thus, on the one hand, we have learned that micro-organisms are always concerned in the inflammation, ulceration, sloughing, and necrosis that occur in appendicitis. The bacillus coli communis appears to be the most common—a bacterium in ordinary circumstances saprophytic and harmless, but which may become virulent and deadly when growing in diseased tissues. More rarely the pus cocci, the streptococcus pyogenes, the staphylococcus, the proteus, and bacillus pyocyaneus are concerned, and the bacillus typhosus has been found to be the organism at fault. On the other hand, we have learnt that peculiarities of position and anatomical formation of the appendix and its mesentery tend to produce contortions and contractions and interference with the circulation in the appendix which prevent the discharge of its secretions, favour the formation of fæcal concretions, and prevent the expulsion of foreign bodies accidentally entering its canal. In one of these ways the vitality of this portion of the intestinal tract, probably owing to its being a vestigial remnant never so high as other parts of the intestine with active functions to perform, becomes impaired. The organisms in the appendix find a medium which enables them to grow and to multiply, often with intense virulence, by which they inflame or destroy the appendix and pass through its walls to the peritoneum and other contiguous parts. Sir Frederick Treves tells us that "it is impossible not to be struck with the fact that perityphlitis runs often in families," not so much as an inherited disease as a family disease. It is much more frequent in the second and third decennia and in the male sex.

So we find the seed and the soil in appendicitis the excitors of active disease and the predisposing conditions—the personal factor

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in this disease—each essential for its occurrence. But whereas in tuberculosis and rheumatism we are often able to detect the mischief by the history, the patient's appearance, the evidence of individual predisposition, there is nothing in appendicitis that reveals a tendency to this condition until the occurrence of an attack of appendicitis. Moreover, whilst the tendency to most of the infective diseases is a general one, more or less distributed throughout the system, though in each there is a favourite mode of entrance of the virus, here it is an entirely local one extending over an area of only a few inches.

Reviewing the group of diseases which I have selected to illustrate my thesis, we have seen that in each there are two factors—the soil and the seed—each necessary to constitute the disease, but that the relative share which they appear to take varies in importance in different diseases. We, as practical physicians, have to obtain our knowledge as to the micro-organisms concerned in these diseases and their chemio-physiological action from those who devote themselves to these studies, whilst we have to observe the effects of the micro-organisms on the human body and how the body reacts to the germs of the disease. The infective diseases existed long before the discovery of the germs, and we have accumulated a large store of knowledge of the clinical course, the treatment, and the issue of these diseases. This accumulated experience is of great value, though apt to be overlooked by the keen interest excited by fresh discoveries. It is against this tendency that I wish to enter a protest, and to point out that what is true is not necessarily new. We must never forget that the *soil* is of as great importance to study as is the *seed* in the proper understanding and treatment of diseases of microbic origin.

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CLINICAL EVENINGS.

November 11*th*, 1901.

CASE OF SYMMETRICAL DISEASE OF THE ANKLE-JOINTS IN A CHILD.

By T. H. Kellock, F.R.C.S.

The patient was a child 3 years and 4 months old, with a history of tuberculosis on mother's side, and the father had had rheumatism in both knees two months before. There was no history of gout or syphilis. There was one other child which was healthy. The patient was breast-fed for one year, and was said to have had rickets, but no other illnesses. The swelling of the ankles was first noticed a year previously; one ankle was affected a little while before the other, and the child had never walked since then. Four months ago the skin broke over both ankles. The swelling on the back of the left hand had not then been noticed. When admitted to the hospital the condition of the joints was much as when shown, enlarged and pulpy, without pain or much tenderness. There were several superficial ulcers round both, discharging a little pus; these had almost entirely healed under quite simple treatment. There was, as now, a fusiform swelling on the back of the carpus of the left hand—this, too, was neither painful nor tender. There was no enlargement of spleen or glands, with the exception of some in the left groin, probably caused by a small abrasion in knee. No signs of tubercle were present in the lungs or other viscera. The temperature was normal, and the child's general health appeared to be good.

The case was probably one of osteo-arthritis.

Mr. Hutchinson, jun., asked why Mr. Kellock believed the case to be a peculiar form of osteo-arthritis, and not a case of tubercular disease. The fact that both ankles were affected was not opposed to the latter diagnosis. It was often the case that the younger the patient attacked with tubercle the slighter was the manifestation. For instance, tubercular disease of the testes would be recovered from in children after existing for months without requiring operative treatment. He would

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like to ask whether Mr. Kellock had ever heard of a case of osteo-arthritis breaking down on both sides, as the present case had done? He would have thought also that the swelling on the back of one hand was confirmatory of tubercular disease rather than of osteo-arthritis. He had had under care a child with many joints and squirmal sheaths affected with the chronic tubercular process, all of which spontaneously subsided, but many of them previously broke down in the way the present case did.

Mr. Kellock, in reply, pointed out that he did mention the fact that there was no history of congenital syphilis in the case, and he could not find any evidence of syphilitic affection about the patient. The point he laid stress on was that the rather extensive ulceration apparently did not communicate with the joints. He attributed the ulceration round the ankles to their having been firmly bandaged for some time, and he thought the fact that the condition got well so rapidly, under simple applications, favoured the view that it was traumatic rather than tubercular.

CASE OF COLITIS WITH PARALYSIS OF ELEVENTH AND TWELFTH NERVES.

By M. S. Monier-Williams, M.A. Oxon., M.R.C.S. Eng., L.R.C.P. Lond.

The patient, aged 56, unmarried, had enjoyed good health until May, 1897, when diarrhœa and hæmorrhage commenced from no apparent cause. The motions were excessively offensive, semi-fluid in character, and occasionally contained, besides blood and mucus, a very little pus. There was no pain except a griping pain when passing motions, which varied in number from five to 15 in 24 hours. There was no rise of temperature, nor was there vomiting. The examination of the abdomen and the rectum revealed nothing abnormal. A course of mercury and iodide of potassium had little or no effect. Rectal irrigations with a solution of nitrate of silver (10 grains to the pint), and of a saturated solution of boric acid held the complaint in check for a time, and a course of sulphur waters at Llandrindod Wells and also at Harrogate benefited the patient for a little, but on the whole he got steadily worse until December, 1900, when he was excessively ill. He had from 10 to 20 hæmorrhagic stools daily. There were extreme emaciation and loss of control of the sphincter ani. Dr. Hale White, in consultation, agreed that right-sided colotomy offered the best chance of saving the patient's life, and Mr. George Turner did the inguinal operation. As soon as the wound was healed, and the artificial

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anus was ultimately fitted with the plug, now exhibited, he rapidly improved. He recovered control over the sphincter ani, and was able to follow his ordinary life without inconvenience. On January 4th, 1899, the same patient suddenly became quite hoarse in his voice, felt a little sick, but not otherwise ill, and noticed that his tongue on protrusion went to the right side. On examination the right vocal cord was found to be paralysed, the tongue was deflected to the right side, and the right side of the palate was partially paralysed. It was considered probable that there was a vascular lesion, probably thrombotic, affecting the nuclei of the hypoglossal and spinal accessory nerves on the right side. The patient had an apical systolic cardiac murmur. Subsequently the upper part of the right trapezius muscle showed signs of wasting, and there was now complete wasting of the right side of the tongue. The patient recovered his voice, but the right vocal cord remained much as before, the left coming over to meet it.

Mr. TURNER said he was asked to do the colotomy first of all with the view to allowing more efficient local treatment to be directed to the colon. He had to bear in mind that if that local treatment were eventually successful it might be advisable subsequently to close the colotomy wound. It was on that account he did not try at the time of operation to make any very definite or complete spur. He did what might be termed the old-fashioned colotomy, not the modern inguinal operation. There might possibly have been some difficulty in making a complete spur as the colon on the right side appeared to be bound down, and he would have had more difficulty in getting it to the surface than was usually the case. It would be appreciated by any surgeon that a colotomy on the right side was a different matter from colotomy on the left, *i.e.*, the material from it must necessarily be more fluid than from an artificial anus on the left side. But he was rather surprised at the exceedingly irritating and offensive matter which came away in the case, and it was at the cost of considerable time and difficulty that Mr. Monier-Williams and the instrument maker devised the plug which was exhibited. Though the patient was previously in a very emaciated and almost moribund condition he had put on flesh, and seemed now to be a different man. It was not for him to speak of the medical diagnosis, but he had privately asked Mr. Monier-Williams whether the consideration of any other case than colitis had entered his mind. Seeing that the patient had had thrombosis about the nucleus of the spinal accessory nerve and hypoglossal nerve, it occurred to him that it was possible there might have been thrombosis of the mesenteric vessels, giving rise to that condition of colon. Twenty years ago he saw a case which had been under the care of the late Sir William Gull, which was published in the 'Medico-Chirurgical Society's Transactions,' in which there was thrombosis of the mesenteric vessels, accompanied by the discharge of blood from the bowel and a slough of mucous membrane, which latter came away almost as the slough of an intussusception. It was, however, composed of mucous membrane. That gentleman entirely recovered, and was now

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well. In his case there was a distinct history of syphilis, and Sir William Gull thought it was a gummatous deposit about the mesentery, giving rise to thrombosis and subsequent sloughing of mucous membrane. He merely quoted the case because of the possibility of this one being not dissimilar. Whatever the condition was he thought the patient and Mr. Monier-Williams were both to be congratulated on the present condition of affairs.

The PRESIDENT said all would agree in echoing what Mr. Turner had said in congratulation of Mr. Monier-Williams in the treatment of the case, and also the patient himself for the improvement which had taken place. Those who had experience of cases of ulcerative colitis, which lasted so long and caused so much trouble, would know the terrible state into which such patients fell; and for the man to improve to the extent he had, and be able once more to carry on his work satisfactorily, and have the discomfort reduced to a minimum by the adaptation of so ingenious a pad to the wound, was a matter of distinct congratulation. In the statement of the case it was mentioned that the cerebral lesion which occurred was due to thrombosis. It was also stated that the patient had mitral disease. It would, therefore, be interesting to know why a thrombus rather than an embolus was suggested. He was unable to see how a plug in the mesenteric vessels of whatever nature could be responsible for the discharge with the peculiar character described which took place after the operation. It was obvious that, until some such plug was used and the material prevented from getting into the lower bowel, the object of the operation had failed; it had not given the opportunity of rest to the colon while the fluid was passing over it. From the history given, and especially from the subsequent course of the patient, he could not think that it was other than exactly corresponded to the ordinary cases of ulcerative colitis.

Dr. BOWLES said he was particularly interested in the association of nervous phenomena with the colitis in the present case. It reminded him of a patient he had seen years ago, in whose case there had been extreme derangement of the nervous system, and at the same time ulceration of the colon. The ulceration spread even externally to the anus, the rectum was involved, and higher up there was evidence of it extending along the upper colon. The patient was very much distressed from spastic rigidity of all the limbs, and was apparently hopelessly ill. He was treated with sour milk and nervine tonics, more particularly with valerianate of zinc. Dr. Bowles thought there was paresis of the viscera as well as spastic rigidity of the limbs. The patient improved, began to be more comfortable, and the limbs became more moveable. In the course of six months he was very much better. Later he was able to walk about, and finally he resumed his position of commander in the Navy. He was so anxious to become perfectly well, however, that, as his back was stiff, he, about two years ago, went to see Dr. Buzzard, who went very thoroughly into his case, and told him that he thought he had suffered from beri beri, and that that had been the cause of his trouble. Dr. Bowles, at the time of the patient's first illness, knew nothing about beri beri, and of course did not treat him for it. Both in that case and in the one now before them, the nervous phenomena were prominent and peculiar, and clearly were part of the general condition which had given rise to the ulceration of the large intestine. Dr. Beevor had pointed out that in the present patient there were peculiarities about the paralysis

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of the tongue; it was, in some ways, different to ordinary one-sided paralysis, and on that account the symptom was a matter of interest, and an encouragement to regard these cases of colitis with associated nerve derangements from a broader and more comprehensive point of view.

Dr. Batty Shaw said he would deal with two points about the case from the point of view of the neurology. The curious weakness in the lower part of the right side of the face he thought suggestive, as proving that the seventh nerve was composite, *i.e.*, derived from the third, the seventh, and the twelfth nuclei. Latterly there had been a good deal of doubt whether the seventh nerve was composite; and he thought this case showed that it was. The second point had reference to the tongue. In the last few months he had seen one other case in which the twelfth nerve on the right side was paralysed. In his own case, which he reported to the Clinical Society, it was caused by a transfacial bullet wound. In both cases the patient had a crinkled tongue on the right side, coupled with wasting there, and when the patient was told to protrude the tongue it came out so that the point was carried to the right, showing weakness of the right side of the tongue. But in both cases the paralysis was not complete, because the patient could, with effort, protrude the tongue towards the left corner of the mouth. He would be glad if any others present could give information on the point, because it suggested that the muscular tissue of the tongue was subserved, not only by the twelfth, but by some other nerve, possibly the spinal accessory, for in both cases the right half of the palate was paralysed.

Mr. Bidwell suggested whether, instead of doing a colotomy on the right side, it would not be desirable to perform an anastomosis between the ileum and the sigmoid. He had made up his mind that in another case he would try that, on account of the trouble patients had suffered from on account of the liquid fæces coming from the right side. He admitted that the ingenious apparatus which Mr. Monier-Williams had devised obviated that in some way, but he did not think the patient's comfort was so great, when wearing a glass apparatus, as it would be if his suggestion were carried out.

Dr. Hale White said he was much interested in Mr. Bidwell's remark. He had himself suggested in conversation with friends that an anastomosis should be done. But the difficulties were two-fold. In the worst cases, and therefore those which would be operated upon, the disease was in the sigmoid. and spread, as in this case and in others, quite below it. He had himself felt, per rectum on more than one occasion, the ulcers in ulcerative colitis. He hoped some day to come to a case in which such short circuiting might be tried, because, as Mr. Bidwell had said, it was clearly the best treatment if one could be sure that the anastomosis would short-circuit the whole of the trouble. Another reason against it was that in many of the cases the gut did not hold very well. The surgeon did not like to do more than he need when the gut was ulcerative. With regard to the question raised by Dr. Bowles, there were two or three cases recorded in the 'Pathological Society's Transactions' which appeared to show that the nervous system might, in some rare cases, influence the gut in the direction of ulceration; and those cases might be regarded in much the same light as Charcot's knee might be looked upon in locomotor ataxy. With regard to the President's remarks, Mr. Monier-Williams

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and he had discussed the possibility of a vascular lesion very thoroughly. It would be impossible to prove the matter one way or the other. He remembered the case to which Mr. Turner referred; in that case the fact that mucous membrane was passed per anum was a great help in diagnosis, but was not present in the case now considered. There had been several fatal cases of thrombosis, and if one might judge by them, the lesion had been altogether more severe than in the present case, the patient dying in a comparatively short time. That seemed to be against the view of there being any vascular obstruction. One thing that struck him about the case was entirely surgical, but it interested him at the time, and he had spoken to Mr. Turner about it. One surgeon who had had a similar operation for the same condition of things preferred a lumbar colotomy, as he got a more dependent opening. In order to prevent the fæces going on to the colon he had seen some surgeons make a much deeper spur. The bigger spur prevented the fæces going on into the colon. In two cases with which he was connected the colon had not been washed out at all from the wound to the anus, and he confessed that they had not taken longer to get well than those in which washing was carried out. He was not sure that perfect rest, with no fæces passing over the part, would not be as good treatment as troubling the patient with washing out. The great question in such cases was the closure, and he might refer to two cases which had been closed and had done very well. One was closed at least five years ago, and when the patient was heard of a short time back she was perfectly well. Another woman, who had to earn her own living, as hospital patients usually did, was operated upon for membranous colitis, the wound was closed, and she had remained well. ever since, a period of three years. She came, within the last month or so, saying she was passing a few shreds again per anum. She was admitted into the hospital and kept there some weeks, but as there was no sign of such occurring she returned to her work. So he thought she must have been mistaken. Having watched the present case, he was convinced that colotomy was not an operation to be done or advised with a light heart; and the same point must have impressed them all. Considering the natural difficulties of a colotomy on the right side, and the fact that possibly the disease might return after the operation, it should only be resorted to in the most serious cases.

CASE OF ACTINOMYCOSIS OF THE BREAST.

By A. Pearce Gould, M.S. Lond., F.R.C.S. Eng.

The patient, an unmarried girl, aged 19, enjoyed good health until the end of August, 1901, when she complained of pain of a stabbing character in the left mammary region. She was examined by a doctor, who thought she was suffering from "intercostal myalgia." A liniment was prescribed and this pain subsided. Nothing further was experienced until five weeks ago when she noticed that the left breast was more prominent than the right; this increased until her admission to Middlesex Hospital on October 29th. The breast was then considerably and uniformly enlarged, fluctuating throughout,

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but neither painful nor tender; the skin over it was not discoloured. An incision was made, and several ounces of slimy green offensive pus escaped. There was imperfect resonance and other evidence of thickening of the pleura. In the pus the mycelium of actinomycosis was found.

All the hitherto recorded cases of actinomycosis of the chest wall, including the mamma, had been secondary to disease of the lungs, pleura, and liver, and this was no exception to that rule. But the case was interesting because of the very slight evidence of the pulmonary disease, and the rather unusual characters of the abscess.

Dr. VOELCKER said Mr. Gould had referred to the fact that he (Dr. Voelcker) had examined the case, and that there was very little to be found on physical examination of the chest, except evidence which pointed to there being some pleural adhesions on the left side. Although Mr. Gould took the view that actinomycosis had originated in the breast itself, it was quite possible that it might have commenced in the pleura. Seven or eight years ago he made a *post-mortem* examination on a case under the care of Dr. Pringle, the subject being a boy of 14 years of age, who had a number of actinomycotic foci in the skin. At the autopsy there was very definite evidence of healed actinomycotic changes in the liver; the pleura on the right side was extensively thickened, and the lung was undergoing extensive fibrosis. Such a case definitely pointed to the fact that an actinomycotic focus might undergo healing in one part and yet might spread in other directions. The existence of pleurisy in the present case, he thought, might make it possible that the trouble had occurred primarily in the pleura, and that the breast had been involved secondarily.

Mr. CARLESS asked whether Mr. Gould could explain why the pus in that instance was so very offensive? Actinomycotic pus, as a rule, was free from smell.

Dr. MARKHAM SKERRITT said he had a case some years ago in the Bristol General Hospital illustrating the way in which an actinomycotic growth would migrate. In that case the first symptoms were in the lung, which physical examination made it plain was affected. This was supported by the character of the expectoration. But as the case went on the signs altered to those indicating infection of the liver, and the condition became typically one of actinomycotic tumour of this organ, as was soon afterwards verified at the autopsy. At that examination the only remain of lung trouble was a band of dense cicatricial tissue uniting the lung and pleura with the diaphragm and the liver. It was the track remaining through which the disease had passed. No evidence of existing disease in the lung could be found.

Mr. PEARCE GOULD, in reply, said he regretted he could not answer the question. The pus was as offensive as that in an appendix abscess. He thought they must recognise that the disease was not yet fully understood. There was good reason to think that there was more than one species of streptothrix involved in the disease. Mr. Foulerton, of the Middlesex Hospital, thought that the fungus in the present case was not the same species as that originally described by Israel. The difficulty

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he had had in cultivating it lent support to that view, and Mr. Gould suggested that the strong odour of the pus was further evidence of the same fact.

AMPUTATION OF THE WHOLE UPPER EXTREMITY FOR CHONDRO-SARCOMA.

By Albert Carless, M.S. Lond., F.R.C.S. Eng.

M. A. B., aged 54 years, was admitted to King's College Hospital, March 13th, 1901. For three or four years previously she had complained of pain in the muscles of the right shoulder, and had been treated for muscular rheumatism. Ten months previously she had noticed a hard swelling below the right clavicle; the pain increased and became shooting in character, extending down to the finger and keeping her awake at night. She went to St. Mary's Hospital, and was treated for chronic rheumatism. Two months before she was seen by Dr. Soltau Fenwick at the London Temperance Hospital, and an operation was advised. When first seen at King's a swelling was present below the right clavicle in the region of the coracoid process. An X-ray examination gave no definite result. When admitted the sub-clavicular swelling was large, and another softer one had developed below the outer portion of the scapular spine. An indefinite mass could be detected in the right axilla, and, on inspection, the whole contour of the shoulder was altered, suggesting a growth of the scapula, enveloping the head of the humerus, which could be felt to be normal in size and position. The movements of the shoulder were limited, especially abduction and rotation, and some crepitus was noticeable in the joint. The muscles of the right arm were atrophied. There was no œdema of the arm, and the radial pulses were equal on the two sides. No glands could be felt either in the axilla or in the supra-clavicular fossa. Urine normal. No cough.

Operation, March 15th, 1901. The whole extremity was removed by Berger's method. The posterior swelling was unintentionally incised, the overlying tissues being thin. Very little blood was lost, and the patient stood the operation well. On March 17th the drainage tube was removed, and on the 23rd the stitches also, the wound having completely healed. On April 3rd the patient left the hospital. The examination of the specimen (which was shown) proved it to be a chondro-sarcoma of the neck of the scapula. There was no sign of recurrence 18 months later.

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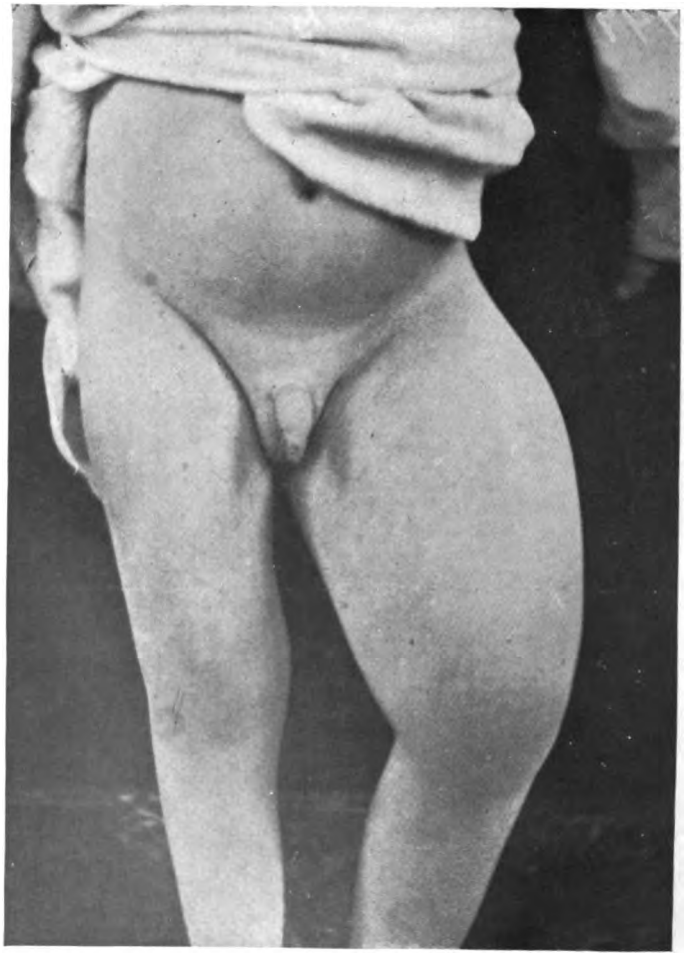

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CASE OF OCCIPITAL MENINGOCELE.

By J. LYNN THOMAS, C.B., F.R.C.S. Eng.

MR. J. LYNN THOMAS showed the photograph of a case of occipital meningocele. The patient was a child, aged 4 months, who had had a tumour at the back of the head since birth which had been getting gradually larger; it was attached to the skull by a pedicle over the posterior fontanelle, and was of the shape of a very large fœtal head.* It was translucent and fluctuating, and had been tapped several times, and it had a number of large veins visible upon its surface when the skin was thin. The child had never been able to move its head on account of the enormous size of the meningocele. Operation was performed on September 24th, 1901. Skin flaps were dissected off the meningocele, leaving the numerous veins untouched. A pair of strong occlusion forceps, six inches long, were then applied to the pedicle and gradually closed, whilst the meningocele was tapped. The pedicle was rendered water-tight by means of a Gely's suture and two rows of continuous sutures. The skin flaps were brought together and collodium dressings were applied. The recovery was uneventful.

TWO CASES.

By W. H. BATTLE, F.R.C.S. Eng.

(a) *Bony Tumour of Thigh.*

THE patient, a boy, aged 4, broke his left thigh in April, 1901. The thigh was put up in plaster-of-Paris; this was removed at the end of three weeks on account of excessive swelling of the thigh, which was said to have been three times the size of the other one. Since that time the femur had united, and now the patient was able to walk about. The swelling was said to be diminishing in size. The left femur was changed into a fusiform bony swelling measuring 15 inches in circumference, twice the size of the left. There was no pain, and the child appeared to be in good health, and got about. There was no softening, and no enlarged glands could be felt.

* Its circumference was 2¾ inches greater than that of the occipito frontal.

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Mr. TURNER asked whether Mr. Battle thought the tumour of the thigh was independent of the fracture? To him (Mr. Turner) it seemed to be simply a mass of callus round the fracture. He did not gather where the fracture was, but he certainly thought it was a case of exaggerated callus following extensive blood extravasation. He had seen a similar condition higher up, about the upper third of the femur, on several occasions. In one such case there was apparently so distinct a tumour that, at a consultation of several eminent surgeons, it was proposed to cut down and explore; some of the surgeons going so far as to recommend amputation at the hip joint, under the supposition that it was sarcoma. However, it got well, and the patient was now walking about. He regretted there was not a skiagram accompanying the patient, as that would certainly have thrown light on the point.

Mr. BATTLE, in reply, said he had no information about the exact point at which the fracture occurred, but surmised it was at about the usual situation of fracture of the femur in children. He could not see the femur properly by means of the fluorescent screen, and was awaiting the X ray photograph which had been taken. He thought that the possibility of it being callus was not a likely one. At one time three possibilities were discussed:—(1) sarcoma, (2) excessive callus, (3) some excessive exudation of blood at the time of the fracture, and some more recent bony formation in that. Possibly, at the time of the injury, the child might have been suffering from some condition similar to scurvy rickets, which was the only way he could account for the extravasation of blood. He had never seen such a large development of callus as this would be (if it was callus), except in some adults suffering from locomotor ataxy. He had had one or two cases under his care in which he was in doubt for some time whether the swelling after a fracture was sarcoma of the femur or excessive callus. In one case he thought for some months that the patient had sarcoma, and that her fracture had occurred in consequence of the sarcomatous change in the bone. But the swelling, instead of getting larger, became smaller. Though it was two years since he first saw the case, the tumour had not yet disappeared.

On the 8th November, 1901, amputation at the hip-joint was performed by antero-posterior skin flaps. The muscles were divided close to the innominate bone. During the operation very little blood was lost, the aorta being compressed by Mr. Corner. There was no shock.

He was discharged on the 18th January, 1902.

For the following microscopic description of the tumour I am indebted to Mr. S. G. Shattock:—

"Half of the thigh and upper part of the leg of a child, in whom the growth of a malignant tumour followed, apparently, upon a fracture of the femur. The shaft of the femur is surrounded by a sarcoma, nearly the whole of which is hardened from osteoid transformation. The growth is most prominent on the anterior aspect, where it is 1½ inches in thickness. A short distance below its middle the femur presents the signs of a repaired fracture. There is no extension of the growth within the medullary canal."

The X rays showed a general uniform osteoid invasion, not radiating, but generally infiltrating.

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(*b*) *Case of Closure of Artificial Anus after Operation for Chronic Obstruction.*

The patient was a man, aged 49 years. He had been in good health until January 1st, 1899, when he felt pain in the lower part of the abdomen, with constipation. About 14 days later he had offensive vomiting and diarrhœa, mucus was passed by the bowel, and he lost flesh. An exploratory incision was made in the median line below the umbilicus. The large intestine was found to be thickened and distended. The upper part of the sigmoid flexure was fixed to the pelvic brim. Below this point a lump could be felt, thought to be malignant growth. There was some adhesion of the small intestine to the swelling. The incision was closed, and a left inguinal colotomy was performed. A month later he was discharged, wearing a colotomy belt. On June 3rd, 1899, he had some return of the symptoms, and the abdomen was reopened in the median line. The growth was found to be smaller than at the previous operation, but still fixed to the pelvic brim. On April 17th, 1901, he was readmitted. Up to Christmas, 1899, he discharged fæces only by the colotomy opening, but about then he began to pass fæces per rectum. The first time he had severe pain in so doing, the pain lasting two hours. Since then fæces had passed both ways. He had greatly improved in health. The abdomen was opened in the median line through the old scar, but no growth or intestinal obstruction was found. On May 30th the colotomy opening was closed, and the patient was discharged on the fortieth day with the wounds almost healed.

TWO CASES OF SNAP OR TRIGGER THUMB.

By Harold L. Barnard, M.S. Lond., F.R.C.S. Eng.

The first case was that of a woman, aged 30 years, upon whom an operation was performed, the patient being perfectly cured. The other case was that of an infant, aged 11 months, the top joint of whose right thumb was sometimes fixed below a rectangular position, and at other times above it. The snap was elicited when the flexed joint was passively extended.

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February 10*th*, 1902.

CASE OF TRAUMATIC RUPTURE OF THE PROSTATIC URETHRA IN A BOY.

By Edmund Owen, F.R.C.S. Eng.

In this case the urethra was ruptured by external injury in an unusual situation. The most common site of rupture, when occasioned by external violence, is certainly the membranous portion of the urethra, but in this case the urethra was torn behind the posterior (superior) layer of the triangular ligament. This lesion is not very rarely produced by fracture of the pelvis, though in the case recorded below no signs of fractured pelvis were present. For the notes of the case Mr. Owen is indebted to Mr. W. J. Finlayson, dresser.

On the afternoon of October 21st, 1901, some children were playing in the timber-yard of an undertaker, when they upset a pile of coffin-lids, which fell upon them, killing one of them. As a result of this accident, a boy, aged 7 years, was brought into St. Mary's Hospital with compound fracture of each tibia, and in a state of serious collapse. The house surgeon, Mr. V. Nesfield, at once took him into the theatre, and, under the influence of chloroform, cut down on to the right tibia, clearing dirt and blood-clot from the wound; then, having sutured the periosteum, which had been extensively stripped up, he inserted a drainage-tube and dressed and fixed the limb. He treated the left tibia in the same way (this fracture was comminuted as well as compound, and the fibula was also broken and exposed). Marks of severe bruising were noticed over the lower part of the abdomen.

When the house surgeon saw the boy at night he found that some blood had escaped from the urethra, but that no urine had been passed. He, therefore, tried to introduce a catheter, but though he could not make it enter the bladder, he drew off a little blood-stained urine. As the boy was recovering somewhat from the shock, and as there did not appear to him to be urgent need or further active interference, Mr. Nesfield decided to leave the boy quiet for the night.

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Next day Mr. Owen saw the boy, and, finding that no urine had been passed, he had him at once taken into the theatre. The bladder was evidently distended, and there was great tenderness all over the hypogastric region. There was slight fulness in the perineum, and some blood was oozing from the meatus unirarius. As it was impossible to pass a catheter, Mr. Owen opened the urethra in the perineum, but he found there neither blood nor urine; and as he still could not hit off the way into the bladder, he had the boy placed flat, and made an incision through the lower part of the linea alba, so as to expose the front of the bladder below the peritoneum. This being done, and before the bladder was reached, blood and urine freely escaped, showing that leakage had occurred into the pelvis. But the bladder was so much distended and was so firm that it was evident that the leakage was not from it. An incision was then made into the bladder, when urine and clots escaped. The pool of stagnant urine on the floor of the pelvis was drained by making an incision through the pelvic fascia and levator ani at the side of the prostate, and passing a large, soft tube through it into the wound already made in the perineum. The bladder and pelvis were then washed out with hot water. The boy was so extremely ill when upon the table that it was impossible to make search for the exact position of the rent; but inasmuch as no instrument could be passed into the bladder, whilst there was escape of urine into the pelvis (but not into the perineum), there could be no doubt but that the rupture was behind the triangular ligament. Had the neck of the bladder itself been torn, the bladder would probably not have remained full of urine under pressure. The rent, therefore, must have been between the neck of the bladder and the triangular ligament. To the surprise of all concerned the boy recovered. For a time some of the urine escaped by the suprapubic wound and some by the perineal drain. Eight days after the operation Mr. Nesfield placed the boy under choloroform for the sake of rearranging the dressings of the legs, and he took that opportunity of exploring the bladder. He then found that he could pass his finger through the suprapubic wound and out through the perineal incision, and he formed the opinion that the rupture was in the prostatic portion of the urethra. With his finger thus in the bladder, he succeeded in introducing a steel bougie through the urethra and into the bladder. Gradually the openings closed and

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the urine flowed more abundantly by the proper route. From time to time a No. 7 English bougie has been passed to give assurance that no contraction is taking place, and up to the present all the urine has come the right way and the boy is convalescent.

TWO CASES.

By WILLIAM EWART, M.D., F.R.C.P.

(a) *Case of Senile Anæmia associated with Gouty Rheumatoid Arthritis and Dyspepsia.*

THE patient, aged 68, commissionaire, was admitted into hospital on November 20th, 1901, with great pallor (without yellowish tinge), debility and slight anasarca (without albuminuria or valvular disease), anorexia and flatulency, with foul tongue and gums, and an indolent, painless rheumatoid swelling and stiffening of both wrists and mid-phalangeal knuckles, and of both knees. This swelling had set in about three weeks previously. No hæmorrhages or any other definite cause could be found for the anæmia. The patient had had syphilis 40 years ago, and gonorrhœa 23 years ago. He also had ague in India. He was laid up 20 years ago for one month with gout in the toe, and has had occasional twinges since. He has always been temperate though not a teetotaler. The blood count gave 2,800,000 red and 60,000 white cells per c.mm., Hb., 60 per cent. On careful physical examination no splenic enlargement or other visceral abnormalities could be detected, nor any glandular swellings, or any other indications of cancer. The case was regarded as one of senile anæmia and arthritis of toxic (alimentary) origin, with leucocytosis.

The line of treatment adopted was (1) at first, rest in bed, (2) sanitation of the mouth and alimentary canal, (3) alterative and tonic medicines, and (4) hygiene and massage. The result was a steady but very gradual and slow improvement, the gastritis and dyspepsia yielding first, and the articular swellings next. The latter have now almost completely disappeared, but the slight anasarca persists, and the man is still very pale, though a pink tinge is perceptible in the lobe of the ear, and the pale complexion has a darker tone (perhaps due to arsenic). He is much stronger and feels considerably better, but he has rather lost than gained

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weight in spite of a liberal diet. No alcohol was administered until the last 10 days, when he was allowed, with apparent advantage, a little whiskey, to which he had been accustomed. The chief medicines employed were magnesia and vegetable stomachics, mercury, arsenic, and iron, in addition to purgatives. The case seems to illustrate the value of a "rotation of drugs"*—most progress having been made since this plan was adopted.

The etiology of the anæmia and leucocytosis in this instance is obscure. Latent carcinoma may be present, although the general improvement renders this improbable, and, moreover, the gastritis and rheumatoid arthritis may be regarded as a sufficient pathological burden. The patient's age and gouty bias may explain the slowness of recovery from the anæmia. The case is exhibited with the purpose of inviting discussion on the subject of "Senile Anæmia" to which little attention is devoted in special treatises on diseases of the blood.

The PRESIDENT said that from the history of Dr. Ewart's case much would depend upon when the condition of anæmia became established, but probably that could not be ascertained. The history seemed to show that the patient had had conditions which were not unlikely to have led to anæmia similar to that which he presented, and his partial recovery under a brief period of treatment would go far to justify the view that that was the cause in his particular case. He presumed the rheumatoid arthritis and the anæmia were accidental coincident conditions. It must be the experience of all to see each of those states quite distinct from the other. He had frequently seen senile anæmia, and quite independent of the state of the mouth. His experience led him to believe that, especially in a man of that age, it did not yield to the ordinary remedies. He had hardly ever seen a case of that kind improve.

(b) *Case of Locomotor Ataxy in a Man.*

The patient, a man aged 45, had syphilis 17 years ago, and when a youth he had received a blow on the head which stunned him. For several years he was periodically brought into St. George's Hospital in agonising pain, and as it could not be explained he was taxed with malingering. The attacks began five years ago, and occurred at rare intervals. They seemed to have been of the nature of cramp and wind in the stomach. Two years ago the patient said the pain was like hot wires being darted through him, and the pain spread towards the base of the thorax, and down into the right lower limb. Sometimes the attacks lasted several days. Last

* *Cf.* 'British Medical Journal,' 1898.

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April he was laid up for three weeks, and said that part of that time he was delirious, but he was not in the hospital then. At the end of December he was suddenly seized with very severe pain, and had morphia injected daily for two weeks. There was also vomiting, which had not been present before. The ataxia began about 18 months ago, and his sight began to fail 12 months ago. There was numbness of the right arm and leg, 16 months ago his bladder became weak, and he had rushing sounds in his head. In January symptoms of paraplegia complicated the ataxia, and he was taken into hospital. The treatment employed was rest in bed and good food, with a mercurial pill nightly, also suprarenal capsule three times a day. The remainder of the treatment was directed to educating and exercising the muscles, in order to improve their nutrition. After two and a half weeks he was much better, and could stand unsupported, even on one foot. It was a question whether by persevering with the treatment a very much better condition might not be produced.

The PRESIDENT asked whether the pains which the patient complained of at first seemed to continue from time to time for some months. He also asked whether Dr. Ewart regarded the pain in the abdominal muscles as an early indication of the tabetic condition. His own experience was that pain in the abdominal muscles would be unusual, and it appeared to have lasted two years before anything else very definite occurred.

Dr. EWART, in reply, said one could not rely upon the man's statements, as they were inconsistent sometimes. But there was loss of knee-jerk and loss of pupillary reflex; therefore if one believed in the connection between lightning pains and ataxia, there was the highest probability that the crises were genuine. With regard to the earlier onset of the lightning pains, he thought that disease in the central nervous system would most probably tell on those mechanisms which were most delicate. And one was led to conceive that the connections with the central nervous system and the sympathetic were of such a delicate order that one might viscerally grow and thrive before learning the use of the muscles, and the muscles of innervation would suffer after the visceral nerves had already felt the effects of the disease.

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CASE OF EXCISION OF A TUBERCULOUS HIP, IN WHICH THE WOUND OF EXCISION WAS CAREFULLY LINED WITH THIERSCH'S GRAFTS

By C. B. KEETLEY, F.R.C.S. Eng.

THE patient was 6½ years old in September, 1900, when her left hip-joint (acetabulum and head of femur) was removed by erasion for tuberculous disease. The tubercle, however, extensively affected the superficial parts (subcutaneous and intermuscular). Therefore, three weeks afterwards, operation was repeated, and the whole wound, *including the deep part leading into the cavity left by erasion of the joint*, lined by Thiersch's grafts. All the deep grafts and most of the superficial grafts adhered, and, from a fortnight after this second operation, what little discharge there was came from the superficial sore. This was further grafted, and has occasionally broken out afresh in small parts, which, however, readily heal. The hip-joint itself has given no more trouble. This has never been drained by any artificial tube. The temperature only twice rose above 99·5° during her stay in hospital, and was generally at or about normal. She wore the extension splint shown.

TWO CASES OF LATE RICKETS.

By E. MUIRHEAD LITTLE, F.R.C.S.

CASE 1.—W. H., aged 17, was admitted to the National Orthopædic Hospital on September 10th, 1900, on account of knock-knee.

History.—His mother states that no deformity was noticed until he was five years old. Four or five years ago he was an out-patient at the Evelina Children's Hospital, and wore instruments for some time. He had always been thin, but no particular notice was taken of any muscular wasting.

State on Admission.—Tall, long-legged, thin, with rounded, prominent forehead, and unusually prominent superior maxillæ. There was little or no subcutaneous fat on the limbs, so that all bony prominences were much accentuated. There was marked fixed lordosis; no paralysis, and no contraction of hip-joints. The great trochanter on each side was in Nelaton's line; the right

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lower limb was markedly rotated out, and there was genu valgum, the leg forming an angle of 145° with the thigh. The tibia and fibula were slightly bent in their upper thirds, with the convexity inwards. On the left side the knee was contracted, and could not be extended beyond 155°. The inner tuberosities of the tibiæ and the lower epiphyses of the tibiæ and fibulæ were enlarged, especially on the right side. The elbows were contracted, and could not be extended beyond 150°. No tendons abnormally tight, and no marks of rickets on the ribs or wrists. Skiagraphs showed the bones of the lower limbs to be unusually pervious to the X-rays, and the epiphyseal lines at the knees were unusually broad and rather irregular.

Macewen's osteotomy was performed on both femurs, and the right tibia and fibula were divided just below the tuberosity of the former. All the bones were soft, and easily penetrated by the osteotome.

Patient left the hospital with the limbs in gypsum on December 28th, 1900. Subsequently he was ordered instruments from the boots to the thighs, with ring catches at the knees, and these he still wears.

CASE 2.—The patient, a girl, aged 13½, was admitted to hospital on September 2nd, 1901, for knock-knee.

History.—Mother states that the child was suckled until she was 12 months old, when she began to walk. No history of delayed dentition. Very healthy until three years of age, when she had slight stomatitis (?), measles, bronchitis, and pneumonia. When four and a half years old she had some bowel complaint and diarrhœa, accompanied by vomiting, which continued at intervals until three years ago. As she improved in health it was noticed that she walked badly, but much deformity was not observed until Christmas, 1900. Mother had never noticed anything unusual about shape of head or forehead. There were four younger and two older children in the family, who were healthy, and not known to have had rickets.

State on Admission.—Plump, healthy looking, very small for age, height 46¾ inches, weight (January, 1902) 53½ lbs. There were bosses on the forehead and on the parietal bones; some teeth were carious; chest well shaped, but there were distinct enlargements of the ends of the eighth and ninth ribs on both sides; spine showed

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lordosis; abdomen slightly distended, and the muscles somewhat flabby. The liver and spleen were normal to palpation. The lower ends of the ulnæ and radii were enlarged, and skiagraphs show cupping of the diaphyses, as in infantile rickets. She had knock-knee, the ankles being 5½ inches apart when the knees were in apposition. Tibiæ not obviously deformed.

On September 11th Macewen's osteotomy was performed on both sides, and the legs straightened and at once put up in gypsum. On October 25th the gypsum was removed, and the ankles could be put in apposition without strain. Discharged November 5th, in long outside wooden splints. Walking well.

Mr. Spencer said it might be a question of name, because very little was known about rickets, or osteomalacia of puberty. The difficulty with regard to late rickets seemed to be that people referred so much to rickets of infancy as if it were purely a question of feeding. If so, the term "late rickets" was not very good, and perhaps it would be better to speak of the cases as osteomalacia, simple softening of bone, because that would give a more general notion that there must be something wrong in the patient's nutrition, but not necessarily with his diet, as was mostly the case in early rickets. He took it that such a case as the girl shown had often been included under the term osteomalacia of puberty, and had been noted as more prevalent in girls. In some bad cases it had been proved to be osteomalacia very similar to that of pregnancy. For instance, there were cases in which women had been subjected to oophorectomy in order to cure a very much more advanced lesion than the girl had.

Dr. Cautley said he thought there was no doubt that the first case was one of late or recrudescent rickets. But he differed strongly from the last speaker in including it under the term osteomalacia. In that condition there was definite enlargement of the epiphyses, and in one case which came under his own observation the epiphyses of the long bones were very extensively enlarged. But whether it was a late condition, and could arise independently of former infantile rickets, he thought extremely doubtful. He was inclined more to the view that such cases were simply recrudescence of the rickets occurring in early infancy. He thought the second case might more definitely be called osteomalacia, or as belonging to a group of that nature, rather than to true rickets. In one point especially that case was very strong—it was entirely limited to the lower limbs, with the exception of some contraction at the elbows. But the softening of the bones occurred in the lower limbs, and on examination there was considerable thickening of the bones of the lower limb on the right side, which one did not see in rickets.

Dr. Belfrage asked whether there was any fault in the general hygiene, the surroundings, or the diet, in either of the cases. It seemed that rickets was generally associated with bad feeding or bad hygiene of some sort. Was one justified in classing as rickets cases in which some serious fault could not be discovered in the matter of hygiene?

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Mr. MUIRHEAD LITTLE, in reply, said the wrists were so very typical of rickets, as in the other case he showed a year or two ago, with cupping of the diaphyses, that he agreed with Dr. Cautley that it certainly was a case of rickets. The other case he was doubtful about, partly on account of the muscular atrophy. He could not trace anything specially defective in the hygiene or surroundings. In the first case the parents were of the small tradesman class, fairly well off. In the other case probably there was bad hygiene. His people were poor, and he worked in a boot factory. Possibly the hygienic surroundings would not be worse than the average of the hospital out-patient class.

THREE CASES OF CHRONIC ŒDEMA.

By W. ESSEX WYNTER, M.D., F.R.C.P.

Dr. W. ESSEX WYNTER showed three cases of chronic œdema:—

CASE 1.—The first patient was a single woman, aged 23 years, who had enjoyed good health till November, 1900, when she developed a cough which lasted through the winter. For the past 10 years the patient had complained of attacks of misty vision lasting one or two minutes, which had lately increased in frequency. Three years ago there was some swelling of the lips and tongue, noticed chiefly on rising, and lasting two months. The swelling of the face and legs appeared to have come on gradually, an to have been notably worse since May, 1901, especially after walking or long standing, the legs being at times so stiff that she could only flex them with difficulty. In the mornings the swelling was softer and less distinct. The swelling of the face was noticed a few months earlier than in the legs, especially about the right eye. The general health appeared to have been unaffected, and there had been no pain. The thoracic and abdominal organs appeared to be normal. The reflexes were brisk. The urine was of a dark amber colour, and was cloudy and acid; its specific gravity was 1020; no albumen or sugar was present. Some improvement was noted in the degree of the swelling of the face and the legs; it varied, but it did not entirely disappear with massage, and subsequently it had been as much as at first. With the disappearance of the hardness pitting could be more easily obtained. The temperature was normal throughout, and the bowels acted regularly. Thyroid extract had been given freely for three months without effect.

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CASE 2.—The second case was that of a girl, aged 17 years, who had had erysipelas seven years ago. The mother stated that from infancy the patient's legs were very "fat." About three months ago the face and hands were noticed to be swollen on rising, and the enlargement of the legs increased. The swelling continued to increase daily, and became more persistent. Recently she had noticed some dyspnœa on exertion and polyuria; otherwise the general health had been good. The face was pale, and was uniformly swollen, the eyes being half closed; the hands also were swollen, but were not distorted in shape; and dimples over the knuckles were well marked. The legs were uniformly enlarged and were very firm, only pitting slightly on hard and sustained pressure, the skin not being white or shiny. The thoracic and abdominal organs appeared to be normal. The urine was pale, clear, alkaline, of specific gravity 1009, and was free from albumen and sugar. The pulse ranged from 60 to 70, and was full and regular. There was a marked depression over the trachea, and the rings could be readily traced, the thyroid isthmus being apparently absent. Thyroid tabloids had been increased from one to three daily. While lying in bed there was at first marked improvement, but on the patient getting about the measurements of the legs had returned nearly to their original dimensions, but were not so hard. The temperature had been fairly normal, and the bowels had acted about once a day. There was an increase of seven pounds in weight in three weeks.

CASE 3.—The third case was that of a female, aged 25 years, who stated that she had never felt brisk and active, but had not had any definite illness. For eight years she had suffered with diarrhœa, the bowels sometimes acting every quarter of an hour, accompanied with pain and prolapsus ani. She had suffered with severe depression. There had been menorrhagia of late, with anæmia. The complexion was of a peculiar tallow-white, and the face was somewhat puffy, with drooping of the lower lids and marked thickening of the upper lip, giving the appearance of a facial diplegia or Erb's paralysis; the muscles, however, retained the power of voluntary movement. The hair was rather thin over the forehead. The patient was full-necked, and her speech was slow and monotonous. Knee-jerks were present. The diarrhœa was checked, but after a month the general condition remained unaltered. Five-grain doses

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of thyroid extract were ordered daily, and rapid improvement ensued.

The PRESIDENT thought the cases were very difficult to explain. The case which Dr. Wynter indicated as the one which had scarcely benefited by considerable quantities of thyroid gland was that in which there was less evidence of absence of the gland than in the other two, and which, on the whole, had the appearance rather of œdema in the ordinary sense. Two of the cases seemed nearer allied to myxœdema than to anything else. It occurred to him at first that the cause was possibly some permanent toxæmia, but that did not appear to be the case.

CASE OF ACTINOMYCOSIS OF THE TONGUE AFTER TREATMENT BY CAUTERISATION AND IODIDE OF POTASSIUM.

By T. H. KELLOCK, F.R.C.S.

THE patient, a mason, aged 18, who had lived all his life in London, was admitted to hospital on November 26th, 1901, with hæmatocele of the spermatic cord, for which an operation was performed on December 4th. On December 31st, two days before he was going to leave the hospital, he drew attention to a swelling on his tongue, which he stated he had noticed that day for the first time. On examination a soft swelling about the size of a small pea was seen on the dorsum of the tongue on the right side, rather yellowish in colour, and a somewhat similar swelling on the right side and slightly under the tongue. It was neither painful nor tender. An incision was made into it, and a little blood and pus let out which contained many yellowish-white granules. These, under the microscope and on cultivation, showed masses of streptothrix. The following day, under an anæsthetic, the swellings were both freely incised, scraped out with a spoon, and then freely treated with the actual cautery. A little sloughing followed this, but it soon cleared up, and the man was discharged from hospital on January 9th, 1902. Two days after the operation he was given a mixture containing eight grains of iodide of potassium, three times a day; two days later this was increased to twelve grains, and later to twenty, which he is still taking. The wounds made in the tongue had healed and left it somewhat scarred, and there was a little swelling near the centre of the tongue which might be recurrence, but looked more like contraction due to the scarring.

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CASE OF NODDING SPASM.

By Bernard E. Potter, M.B. Lond.

The patient, a child aged 9½ months, had suffered from nodding movements for four months. The child had always lived in very dark rooms, with the exception of the two months of August and September, 1901, when the parents took her away with them to Norfolk. They returned with her about the middle of September, and the nodding movements began a month later, the child being then five and a half months old. Teething began at the age of five months. The child had been breast-fed, and had had no other ailment. She was well nourished, and had never had convulsions. The mother's labour had been natural, and no instruments were used. The movements were almost entirely in the vertical direction. Soon after these head movements began, a rotatory nystagmus in both eyes was noticed. The interest of the case lay in the following facts—that the movements began almost immediately after the commencement of dentition; that the change from the light rooms in Norfolk to the present dark ones in London appeared to have influenced the child; and that the movements were nodding in character, and not from side to side, as is more commonly the case.

Dr. Voelcker said a point of interest in the case was that it exemplified a feature which was brought out by Dr. Thompson, of Edinburgh, that in those cases the nystagmus differed from that seen in adults—for instance, in disseminated sclerosis. The nystagmus was very often of a convergent type, and on looking at the child shown, he noticed a convergent nystagmus instead of a parallelism of the axes of the two eyes being maintained, which was the case in the commonly-met-with form of nystagmus. Again, although the nodding movements were vertical, the nystagmus was more or less horizontal. There also Dr. Thompson had pointed out that in cases of nystagmus associated with spasm the nystagmus was neither constant nor truly horizontal. And in those cases in which it was a rotatory nystagmus, it did not take place around a point in the centre of the pupil, but in an irregular elliptical form. In the case shown, he could verify the point as to the convergent nystagmus, and that it was constantly in the horizontal direction.

Dr. Cautley said the movements were much more of the true type of nodding than was usually the case. The more common type was where the movements were of a lateral nature, spasmodic contraction of one sterno-mastoid. It was extremely difficult to ascertain the exact cause

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of the condition. He thought dentition could be excluded; the affection occurred in many children long before the period of dentition. Living in dark rooms could also be excluded, as it would occur in children who did not live in dark rooms, and also in many members of the same family who had been brought up in the most advantageous conditions. Rickets had been associated with it, but that must be excluded, because many of the subjects were not rickety. It had also been said that these patients were precocious and had over-strained, but in some cases the nodding movements began before the nystagmus. Sometimes the nystagmus was the only condition present. In the present case the child was rather micro-cephalic, a point which was important from the point of view of prognosis. In cases of micro-cephaly his experience was that the forecast was grave.

CASE OF SCIRRHUS OR ATROPHIC CANCER OF THE TONGUE.

By WALTER G. SPENCER, M.S., F.R.C.S.

THE patient was a thin man, aged 72. He came to the hospital in November, 1901, complaining of increasing difficulty in swallowing and speaking for some three months, owing to fixation of the tongue. Since then he had been taking iodide of potassium and mercury, but the disease had progressed, and he required now 10 grains of pil. saponis co. every night to obtain sleep. The patient spoke and swallowed with difficulty. The tongue was seen depressed to the level of the floor of the mouth and fixed, more especially as regards its right anterior half, whilst the epithelial surface was unaltered, except as regards puckering, and there was no evidence of previous buccal inflammation or ulceration. The tongue felt like a board, and formed the surface of a densely hard, not tender, mass to be felt bulging below the jaw, and fixed to the horizontal rami, also the sub-maxillary glands appeared to be already involved in the tumour. The case appeared to be one of the rare variety of malignant disease of the tongue, called scirrhus, or atrophic cancer, from its likeness to that form of disease in the breast, and was mentioned by Butlin, also by Lagoutte and Wright (*see* Butlin and Spencer, 'Diseases of the Tongue,' pp. 322, 462). There was an absence of any history or sign of syphilis, and the antisyphilitic treatment had failed. Actinomycosis of the tongue was attended, in man, by suppuration; when of the fibrous variety, in cattle, it was hypertrophic. It did not appear feasible to attempt

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removal in this patient, so that it was proposed to continue the present administration of iodide of potassium and opium.

NOTE.—*October*, 1902. The patient has been continuously under observation, taking, on the average, 15 grains of pil. saponis co. every night, and now and again a pill during the daytime. The pain is at times severe. The disease, on the whole, remains stationary; at one period the deep cervical glands enlarged, but later shrunk and softened, as if becoming fatty. Similar slighter variations have occurred in the main tumour, but there is no sign of breaking down or of fungation, either in or outside the mouth. The patient is rather weaker, is subject to fainting fits, and the pain often makes swallowing difficult.

TWO CASES OF BELL'S PARALYSIS.

By HAROLD L. BARNARD, M.S., F.R.C.S.

CASE 1.—The patient, a child aged 2½ years, had the left ear-drum irregular and scarred, and there were dried crusts in the left ear; but there was no history of discharge from the ear, and no sign of mastoid disease. No perforation could be seen. Left Bell's paralysis came on in one night and was complete. There was an obscure history of a slight fall seven days before the onset.

CASE 2.—The patient, a boy aged 4½ years, had a severe fall on the left side of the head, and was stunned, but recovered. Three days later he became feverish, delirious, and unconscious. Three days later he recovered, and it was found that he had left facial paralysis and right hemiplegia not involving the face. The hemiplegia rapidly cleared up, and when Mr. Barnard saw him, two months later, he was able to move the right arm and leg well, but the tendon reflexes on the right side had increased. When shown there was no appreciable difference between the sides.

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April 14*th*, 1902.

INFANTILE FORM OF ARTICULAR RHEUMATISM, WITH CHRONIC ENLARGEMENT OF THE SPLEEN, LIVER, AND LYMPHATIC GLANDS.

By F. Parkes Weber, M.D., F.R.C.P.

The patient, Arthur S., a rather pale boy aged 4¾ years, was regarded as a typical example of an affection which has been well described by Dr. G. F. Still,* and was usually classed as a variety of rheumatoid arthritis in children. In the present case there was some apparent enlargement of the knee and elbow joints, but the most marked change was the symmetrical swelling about the articulations of the hands and feet. The swelling, as investigated by the exploring syringe and Röntgen photographs, seemed to be due to infiltration into the periarticular tissues, with little, if any, effusion into the synovial cavities. In the feet it was most marked over the metatarsus, in the hands over the proximal ends of the metacarpal bones and over the finger joints. There was considerable stiffness in the cervical spinal column, and this accounted for the extreme movements of the eyeballs which often give the face a remarkable appearance, such as that seen in the accompanying illustration. The heart was enlarged. The apex beat was in the fifth space, one inch outside the nipple line (April 7th), and the first sound at the apex was replaced by a musical murmur. The spleen and liver extended considerably below the ribs, and the lymphatic glands in the neck, axillæ, and groins were enlarged, but not greatly so. The temperature had not reached 100° F. since February 3rd, and, of late, the child had had a good appetite, had appeared to put on flesh, and had seemed happy and free from pain and tenderness in the joints. The urine contained no albumen or sugar. A blood count of January 26th gave 4,600,000 red and 31,000 white corpuscles in the cubic millimeter.

From November to December, 1900, the boy had an acute disease, said to have been articular rheumatism. Before this he

* *See* Dr. Still's account of rheumatoid arthritis in children in Allbutt's 'System of Medicine,' 1897, vol. iii, p. 102; also his paper read before the Royal Medical and Chirurgical Society in 1896.

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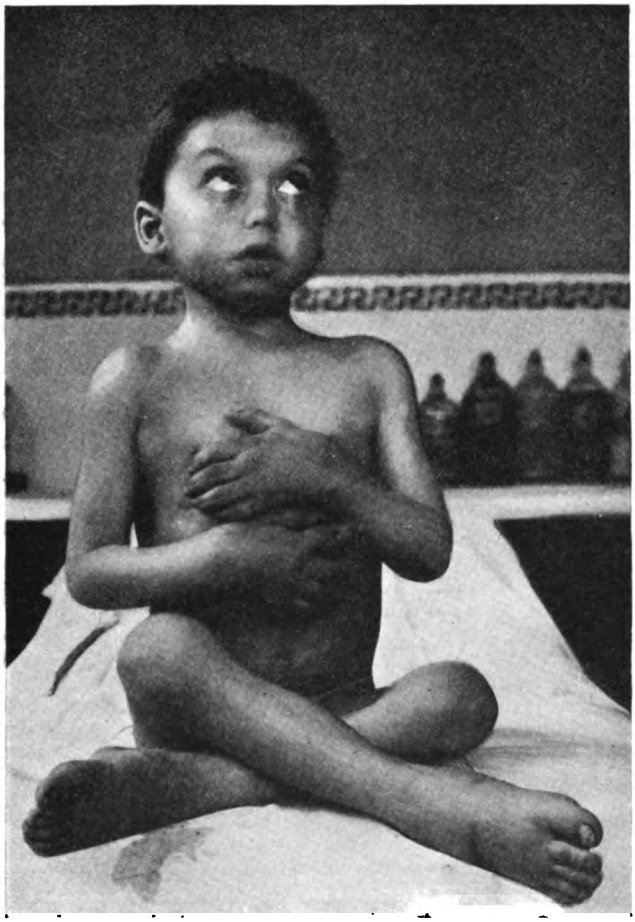
could walk, and was apparently healthy. The cervical rigidity had lasted since Christmas, 1900. At one time it gave rise to a suspicion of spinal caries. The swelling of the hands was noticed a short time before May 1st, 1901, when he was admitted to the



German Hospital. The articular affection of the feet was little marked on admission, but became a decided feature of the case by the end of August, 1901. Under treatment by rest, aspirin, iodide of iron, arsenic, malt, &c., the boy's condition had fluctuated

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considerably in regard to swelling and tenderness of joints and general appearance, but wasting of the muscles of the limbs had progressed. Occasionally there had been moderate fever, and about the beginning of November, 1901, there was a definite attack of endocarditis, and the heart, which previously had appeared healthy, or only very slightly dilated, showed signs of disease of the mitral valve.

It was this attack of endocarditis, and the fact of pericardial adhesions having been found in other cases, which made Dr. Parkes Weber think that the affection was more allied to articular rheumatism than to any forms of rheumatoid arthritis in adults. Supposing the ordinary cases of acute and subacute rheumatism to be due to a specific microbic affection, as the recent work of Dr. F. J. Poynton and Dr. A. Paine made probable, cases like the present one might perhaps be due to the same specific cause, the clinical differences being explained as modifications resulting from the age of the patients. In the same way tuberculosis in young children is, clinically, generally very different from tuberculosis in adults. With the consent of the parents Dr. Weber asked Drs. Poynton and Paine to try if they could obtain microbic cultivations from the joints. This they kindly attempted in the commencement of the present year, but the attempt failed, the exudation apparently being gelatinous and in the periarticular tissues. There was little, if any, effusion into the cavities of the joints.

Dr. A. E. Sansom said he could only regard the case either as one of rheumatism, plus something else, or as not being rheumatism at all. He did not regard the arthritis as one of rheumatic origin, the characters of which were a relatively painless effusion into the joints of a few weeks' duration. The cardiac lesion in this case was very slight in his opinion, if, indeed, any existed. There were present osteo-arthritis, marked atrophic nerve phenomena with enlargement of the liver and spleen, a general dyscrasia, but nothing whatever of rheumatism. In his belief the cases described by Dr. Still were in no way connected with rheumatism.

Dr. Cautley regarded the case not as one of rheumatism, but as one of osteo-arthritis occurring in a young and rachitic child, for the child seemed to have had rickets, and the enlargement of the liver and spleen was not more than that sometimes seen in rickety children. With regard to the view that the associated symptoms made the case a special disease he was inclined to doubt it, for the reasons that there was often a moderate enlargement of the spleen in children who had any febrile condition, and in osteo-arthritic cases there was considerable fever,

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sometimes long continued. Enlargement of the glands in children was so common, and due to such slight causes, that its association was not of any importance. There was, undoubtedly, a cardiac murmur with some hypertrophy of the heart, and he did not know of any reason why the child should not have had endocarditis in addition. He had seen osteo-arthritis in one member of a family, rheumatic fever in another member, and chorea in another.

Dr. ROLLESTON regarded the case as belonging to the category described by Dr. Still, but it differed from ordinary osteo-arthritis, inasmuch as the joint changes were chiefly periarticular. It was probably some form of general infection, sometimes the kidneys became affected, and uræmia might prove fatal. Probably the cardiac affection was due to the same cause as the periarticular and glandular changes, and need not be regarded as evidence of the rheumatic infection or alleging it with rheumatic fever.

Dr. ALEXANDER MORISON agreed with Dr. Sansom that there was no definite evidence of organic heart disease in the case. There was a bruit and displacement of the apex outwards, and an apparent increase in the area of the heart. But the rachitic state of the chest would explain those. He agreed also with Dr. Cautley that whatever the arthritic condition was it was grafted upon rickets. The bruit was most perceptible with the child lying down. He regarded it as a hæmic bruit.

Dr. POYNTON remarked upon the exceedingly interesting nature of the case, which he also recognised as one of the type described by Dr. G. F. Still. He had been very glad to have an opportunity of making a bacteriological investigation with Dr. Paine, though the result had been negative.

Were these rheumatic or rheumatoid in character? The answer, he thought, was a difficult one, and be would give a very brief account of two cases bearing upon these questions:—

No. 1.—A boy, aged $4\frac{8}{12}$, whose mother had suffered from rheumatic fever in January, 1901, had an attack of arthritis of the knees and ankles, from which he apparently quite recovered. In March he had arthritis of the left shoulder; in May a third attack; and finally, at the end of July, a fourth attack. When first seen in this last attack he had been laid up a week with fever and arthritis. The temperature was irregular, ranging about 100°. The cardiac area was enlarged, but there was no cardiac murmur. Both wrist joints and both knee joints were swollen, and while under observation an interphalangeal joint of the second toe of the right foot and of the middle finger of the left hand swelled and became red. The swelling was chiefly periarticular. Over the region of the spleen the area of dulness was considerably increased, but the edge was not felt below the costal arch. The case went from bad to worse, and the boy was then lost sight of. He believed this to be a case of the kind under discussion, and if so, it was interesting that there was a family history of rheumatic fever, and that previous to the progressive illness there had been attacks of arthritis which had passed off.

The second case was that of a little girl, aged $4\frac{7}{12}$. Seventeen weeks previously she had suffered from "rheumatic fever." The elbows, knees, and hands were swollen, but the doctor in attendance thought the heart was not affected. He himself had seen the case, because, though 17 weeks

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had elapsed since the commencement of the illness, the knee joints contained a great deal of fluid, the temperature had remained irregular, and a small nodule apparently fixed to the lower end of the left radius had appeared. The nodule resembled those seen in rheumatoid arthritis rather than in rheumatic fever. In this case there was no marked periarticular swelling; no enlargement of the lymphatic glands or spleen. Treatment with salicylate of soda, which had been pushed, was quite ineffectual. Perfect recovery resulted with liberal diet, tonic doses of quinine, extension, and Scott's dressing.

Though this case was clearly somewhat different in nature from the former, it, he thought, demonstrated how cautious it was needful to be in excluding rheumatism. As for rheumatoid arthritis, that disease was at present in a condition of chaos, but he believed that rheumatic fever was one of its causes, such cases being examples of malignant rheumatic arthritis, comparable to malignant rheumatic endocarditis.

Dr. PAINE said that, although the attempt at cultivation of organisms from the joint was negative, such a result was only to be anticipated, because in arthritis, where there was only a small amount of clear exudation, the fluid was generally sterile. The explanation was that the organisms were in the tissues, and so long as the endothelium remained intact there were no organisms in the exudation. But one saw cases of non-septic arthritis in which there were vast numbers of organisms found in the exudation. In those cases probably the endothelium had been destroyed, and by that means the micro-organisms could gain entrance into the synovial cavity. It was interesting that the character of the arthritis resulting from the inoculation of rabbits with the diplococcus of rheumatic fever was identical in its nature with the arthritis met with in the present class of case—*i.e.*, a large amount of gelatinous swelling of the periarticular tissues, with very little exudation in the joint. That would point to an infective origin in such cases.

The PRESIDENT fully joined with those who deprecated the word "rheumatic" in reference to the present case, though he considered there was some valvular trouble in the heart. The physical signs pointed to that. The point of the case was the possible association with acute rheumatism. Dr. Weber suggested that the circumstance of age might have been such as to have influenced the development of the disease, as to have led to the condition presented by the patient, just as the manifestations of tuberculosis might vary at different ages. If so, was not that going too far? Acute rheumatism was extremely common, whereas the condition presented by the child now shown was very rare, and if acute rheumatism usually assumed such a form, the condition might be expected much more frequently than was the case. Moreover, was it not quite possible that the special form of periarthritis which existed in the child might not be quite independent of acute rheumatism, although this latter had supervened?

Dr. PARKES WEBER, in reply, agreed that murmurs of the kind presented by the boy were not necessarily due to organic disease of the heart valves. But in *post-mortem* examinations on fatal cases of chorea vegetations were sometimes found on the cardiac valves when the symptoms of endocarditis observed during life had been even less marked than those in the present patient. When the boy was first seen the heart appeared normal, and the murmur developed afterwards together

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with displacement of the apex beat outwards. Therefore he did not feel inclined to alter his view that there had been an attack of endocarditis. With regard to rheumatism and rheumatoid arthritis, he was on very uncertain ground. He was inclined to think that there were different kinds of rheumatoid arthritis, but, apart from the so-called "pseudo-rheumatisms" due to gonorrhœa, &c., only one form of rheumatism. He did not see any reason for regarding his case as one of septic pseudo-rheumatism of any kind. There had never been albuminuria. No doubt the child was somewhat rickety, but rickets was so common—perhaps one in three hospital children was somewhat rickety—that it did not help to explain the case. Dr. Cautley's suggestion that the case was one of osteo arthropathy in a rickety subject was necessarily true. He acknowledged the justice of the President's criticism, that very young children did get rheumatic fever without this type of joint affection, but then they frequently had rheumatism (shown by endocarditis) without any severe joint symptoms at all. The present type of arthritis might, therefore, represent a relatively rare severe form of joint rheumatism in infants and very young children.

CASE OF CHRONIC NEPHRITIS TREATED ON THE RESTORATIVE METHOD BY DRAINAGE AND FEEDING.

By William Ewart, M.D., F.R.C.P.

A. P., aged 31, single, cook, temperate and otherwise healthy, had suffered for 12 months from recurring attacks of renal œdema. No definite cause for the nephritis could be made out. When admitted to St. George's Hospital on August 3rd, 1901, there was moderate urinary output, 30 to 40 ounces, specific gravity 1010, and albuminuria 1·45 per cent., with slight puffiness and anasarca. The pulse was rather tense, the breath heavy; there was debility, anorexia, malaise, and some headache. Milk diet and purgatives were prescribed with some general improvement, but no great change in the albuminuria. Towards the end of August, whilst still in bed, she had another unexplained aggravation of the œdema which became considerable, and the "drainage and feeding treatment" being now available, both feet were incised on August 31st, and a liberal diet was allowed. On December 5th the right leg was again incised at the ankle, as the previous incisions had, a few days before, ceased to run. The usual improvement was obtained as regards general strength, appetite, and digestion. The albuminuria was lessened, but did not disappear. The pulse became softer, and the body weight and the complexion improved. Massage was subsequently added to the treatment, and she was allowed to walk about

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the wards, fluid still discharging from the incisions. Importance attaches to the case in conection with the long persistence of the œdema, and the long duration of the drainage from the legs (from August 31st, 1901, to January 14th, 1902—a period of four and a half months). During that time, whilst the kidney function remained inadequate, as clearly shown by the persistent œdema, the patient was steadily improving instead of degenerating; and it is not improbable that the nutrition of the kidney may also have improved. At any rate she was at present much better, instead of worse, than six months before, and better able to meet the strain of a fresh attack if it should occur. She was still able to take the same liberal mixed diet; and this was encouraging, Dr. Ewart's experience being that where little secreting substance remains, a generous diet quickly leads to uræmic symptoms after the vicarious relief of artificial drainage has ceased. The albuminuria persisted (about 0·6 per cent.), and there was slight œdema of the legs which fluctuated in its degree. She was taking in rotation mercury with iron, arsenic with strychnine, and an alkaline stomachic with nux vomica, and she was looking better, was gaining strength, and declared that she was feeling quite well.

The principles of this treatment were first published four years ago before the Royal Medical and Chirurgical Society,* where they were somewhat adversely criticised. The patients submitted to it are conscious of its value. Prior to its publication others, including Dr. Hale White,† had advocated a mixed dietary. Dr. Ewart believed that in bad cases this could not succeed without the protection afforded by drainage. Unfortunately the benefits of the treatment which he had described are limited to those cases in which chronic nephritis is accompanied with anasarca.

The PRESIDENT asked whether Dr. Ewart thought the method of drainage described had any great advantage over frequent sweatings, as the latter rapidly brought down œdema in a large number of cases. He agreed with him that in such cases there was an increase in the urine secreted, when the skin had been brought to act freely, or the anasarca had been relieved by drainage.

Dr. SANSOM believed that there was chronic parenchymatous nephritis in the case, and the treatment was very successful. In a case at the London Hospital, without much œdema, but with symptoms of uræmia beginning, the feet were punctured, the patient propped up carefully with

* *Cf.* 'Proc. Roy. Med. Chir. Soc.,' 1898.

† *Cf.* 'Trans. Roy. Med. Chir. Soc.,' 1893.

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pillows, and his feet put into a warm footbath. That was done for several days, the fluid draining into the bath. The water of that bath became ammoniacal to a degree. The patient ultimately lost all signs of the uræmia.

Dr. EWART, in reply, said that he had tried the treatment by sweating with considerable temporary benefit in many cases, but that the results had not been permanently satisfactory. One reason for preferring the method which he had described seemed to him to be that it at once and on a large scale removed effete products which were lodged in the tissues from the danger of being taken into the circulation. One never knew how much poisonous material might remain in the tissues after sweating had been carried out. There was also another practical point in favour of this treatment: it enabled the patient to take and relish an abundance of varied food, and to digest and assimilate it, and this was an important therapeutical indication in all diseases, and particularly in Bright's disease. The weak point in the treatment was that it could not be applied in some cases where its advantages were most desirable. It could only be used where fluid collected largely in the lower extremity. In some of the worst cases fluid would not collect. It could not claim to be regarded as a radical treatment dealing with the cause, but merely as an effectual remedy for some of the worst results of the disease.

THREE CASES.

By BERTRAND DAWSON, M.D. Lond.

CASE I.

Fibrosis of Lung, probably Syphilitic.

C. K., a woman aged 50; five children all healthy, no miscarriages. No previous illness of importance till the present one. No history of secondary syphilis. Twelve years ago a lump came on the right shoulder: this lump became an "abscess," and then burst.

The onset of the present illness was gradual, beginning nine years ago with a hacking cough, which did not, however, at first affect her health. Eight years ago she became somewhat suddenly ill, was stiff and tender all over, and thought she had rheumatism. A few days later she called in a doctor, who, after examining her, said she was in consumption. She had at that time been ill for three months with cough, spitting of phlegm, frequent slight hæmoptysis (not more than a drachm at a time), and night sweats; the amount of sputum was not large in quantity, and it was not fœtid. From then till six years ago—that is, for a period of two years—she was continuously ill with the same symptoms.

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Six years ago Dr. Dawson saw the patient for the first time. There were the above symptoms of sweating, cough, spitting, and slight hæmoptysis. There was slight and irregular fever, and the fingers were clubbed. The physical signs were confined to the right chest, and these could have been regarded as an active supervening on a chronic phthisis, with signs of cavitation, the latter most marked at the posterior base. The left lung was free. The sputum was muco-purulent, was sometimes fœtid, rather large in quantity, but contained no tubercle bacilli.

Dr. Dawson believed at the time that the right lung was the seat of some non-tubercular fibrosis, with perhaps some dilatation of the bronchial tubes. At this time a lump appeared in the right sterno mastoid muscle, and another the size of a walnut over the ascending ramus of the left jaw close to the articulation, and involving the pinna of the ear. Very shortly afterwards yet another swelling, the size of an egg, appeared over the right shoulder. These swellings were kindly examined and treated by Mr. John Murray, Assistant Surgeon to the Middlesex Hospital. The patient was then put upon iodide of potassium, and the swellings rapidly disappeared, healed, and the chest symptoms markedly improved, as far as the acute symptoms were concerned.

Since the course of iodide of potassium the patient had been in better health altogether; the hæmoptysis and sweating had ceased; her weight had remained constant, she was able to get about, and in the summer time her cough only troubled her on rising in the morning. She is of necessity, however, a pulmonary invalid, for the fibrosis of the lung persists. One point of interest is that, since the subsidence of the active mischief, the sputum is more often brought up in large quantities at a time, and is oftener fœtid. A specimen of the sputum was shown at the meeting. The case had assumed more of the features of a bronchiectasis.

The case was, without doubt, one of non-tubercular fibrosis of the lung, with some secondary bronchiectasis. There was a natural hesitancy to diagnose syphilis of the lung on account of its rarity, and of the difficulty of excluding other causes of pulmonary fibrosis. The facts, however, in this case were strongly in favour of syphilis, because, first, the acute symptoms and signs in the chest ameliorated under iodide of potassium; secondly, the symptoms and signs especially characteristic of fibrosis came on subsequently to the anti-syphilitic treatment—that is, as the result of the healing of

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some active process; thirdly, that active process was not tubercle, though its manifestations in part resembled those of tubercle—there was no history of pneumonia, which often explained unilateral fibrosis; and, fourthly, there were evidences of tertiary syphilis antecedent to the chest mischief, and there were definite signs of syphilis concurrently with the active lung trouble and healing simultaneously with it.*

Dr. VOELCKER said the case was a very good example of syphilitic affection of the lung. Clubbing of the fingers was very marked. But he suggested another explanation for the condition, which he took to be one of bronchiectasis, and that instead of supposing there was a diffuse fibroid of the lung producing traction on the bronchi and so causing bronchiectasis, it was probable that there was a gumma in the main bronchi. Some years ago he had an opportunity of making a *post-mortem* examination in a case in which there was evidence of one particular healed and cicatrised gumma in the left bronchus, and in that position there had been produced marked stenosis of the bronchus, and, secondarily to that, bronchiectasis occurred. Bronchiectasis arising from obstruction to the bronchus was seen not only in syphilis, but also where there was narrowing of the bronchus from either a malignant growth or from an aneurysm pressing on the bronchus. He suggested that in the present case, especially as the symptoms developed soon after the fortunate and successful treatment with iodide of potassium, one was dealing with a healing gumma in the bronchus, rather than with a primary diffuse syphilitic fibrosis of the lung.

Dr. ROLLESTON suggested that the increased secretion of mucus noticed after the administration of iodides was perhaps due to the action of the drug. Some years ago, in conjunction with Dr. Cyril Ogle, he had published a case in the 'Transactions of the Clinical Society,' vol. xxxii, p. 158, of a young woman, who died with syphilitic stricture of the bronchi, in whom the administration of iodides greatly increased the amount of secretion retained in the bronchial tubes distal to the obstruction. There was thus considerable danger in giving iodides to patients with stricture of the bronchi, or where an aneurysm pressed on the bronchi. When this drug was given in such cases it should always be combined with, and guarded by, full doses of belladonna, which counteracted the increased secretion induced by iodides.

Dr. DAWSON, in reply, thought Dr. Voelcker's suggestion quite as reasonable as his own. He (Dr. Dawson) did a *post-mortem* on a case of aneurysm pressing on one of the larger bronchi, in which the area of lung supplied by that bronchus showed the most beautiful specimen of dilated tubes he had ever seen. With regard to the action of the iodide, the bronchiectatic character of the case was of slow development after the iodide. The patient also had from time to time vomited up fœtid sputum.

* This patient died unexpectedly of hæmoptysis about a month after the meeting. The *post-mortem* disclosed a specimen of syphilitic lungs.

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Case II.

Aortic Incompetency, with well-marked Diastolic Thrill widely felt over the Præcordium.

The patient, a man aged 22, came to hospital in December, 1901, because he was breathless, and had at times a dull pain over the præcordium while at work, but did not suspect that anything was the matter. No history of any previous illness. Physical signs:—The præcordium was bulged. Apex beat felt in the sixth space and in the nipple line. The impulse was heaving and followed by collapse. At the apex there was no thrill. The area of cardiac dulness increased down and out, but not either upwards or to the right (*see* figure). A thrill, diastolic in time, was felt over a wide area of the heart's base, this area being represented on the diagram by interrupted lines, and the point of maximum thrill marked by a cross, thus +. The area of the thrill was seen to extend upwards to the upper border of the third rib; it was not dull, except where it corresponded with the normal area of cardiac dulness. There was no expansile pulsation. At the apex beat the first sound was fairly clear, and was followed by a diastolic bruit occupying the first part of the pause; the bruit was heard outwards as far as the mid axilla and over the back. Over the base of the heart the first sound was very short and indistinct, and was followed by a diastolic bruit, harsh in character, and occupying the *whole* of the period of the pause, only ceasing with the subsequent first sound. The point of maximum intensity of the diastolic bruit was the same as that of maximum thrill, and is indicated by a +.

The pulse was very collapsing. There were no pressure signs of any kind. The skiagram shows neither evidence of saccular aneurysm nor of aortic dilatation.

The existence of the aortic incompetence hardly called for discussion. The interest of the case centred in the explanation of the loud diastolic thrill and the point of maximum intensity of the bruit.

Was there an aneurysm present? Dr. Dawson thought not. There was no expansile pulsation and no dulness, and if an aneurysm were near enough to the surface to give so well-marked a thrill it would be near enough to give dulness and expansile pulsation. The fact of the thrill, being diastolic was an unusual

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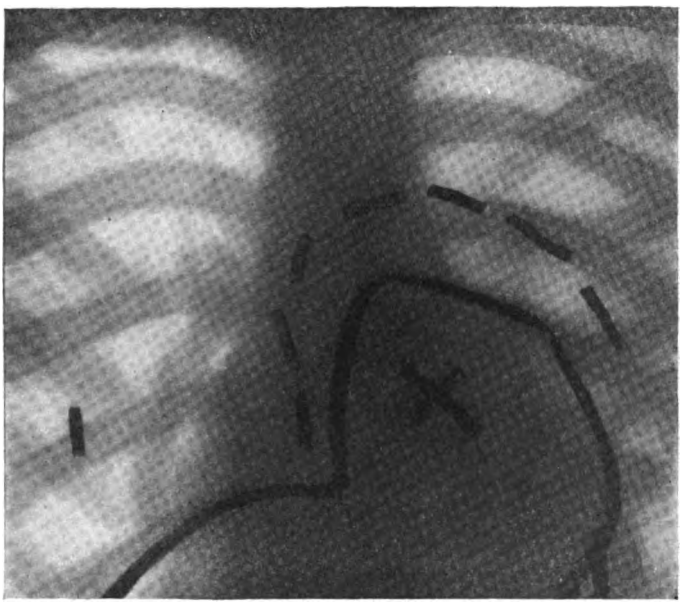
but not unknown feature of an aneurysm. Further, there were no pressure signs and no tugging, and the radiograph disclosed no sac or aortic bulging. The point of maximum intensity of the bruit was shown to be over the heart itself. He suggested that the phenomena were best explained by supposing the existence of either a perforation in one of the injured cusps, or of a rigid calcareous valve projecting into the blood-stream. The fact that the bruit at the point of maximum intensity endured throughout the *whole* of the diastole suggested rather a flow of blood through a narrow opening than such as a perforation.



He had once done a *post-mortem* on an aortic case in which there had been a diastolic thrill. The aortic valves were calcified stumps, and the aortic orifice was consequently of very irregular contour. It was clear that the cause of such a bruit must be determined not only by the size of the orifice, but also by the shape of the latter, and the character of the tissue which bounded it.

Dr. WEBER asked whether in this patient there could be septic endocarditis of the pulmonary valves. He had seen a case with a somewhat similar diastolic murmur in which there was disease, as shown by the necropsy of the pulmonary artery, probably due to gonorrhœa.

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Dr. MORISON said he had never known so pronounced a diastolic thrill in a purely aortic case. The thrill distinctly marked out the area which one sometimes found the bruit of aortic regurgitation to be limited to. He likewise spoke of the diagnosis between the diastolic thrill in question and that associated with regurgitation through the pulmonary valves. Of the latter he had observed and recorded a good example.

Dr. DAWSON, in reply, said the pulse was typically collapsing, which was strong evidence of the lesion being aortic. It was too low down to have anything to do with the pulmonary valve. Moreover, it was diastolic, and its maximum intensity was nearly at the fourth cartilage.

CASE III.

Phthisis in Conjunction with Albuminuria and Paralysis of the Left Sixth and Seventh Cranial Nerves.

The patient, a man, aged 27, has had gonorrhœa, but not syphilis to his knowledge. In 1890 had tubercular disease of right knee, which was cured in hospital a year or two later. His present illness began about March, 1899, with a cough, which had persisted on and off ever since. There had been wasting and night sweats from time to time, but never any hæmoptysis. In February, 1901, he went to Ventnor for 10 weeks, and gained 1½ stone. His present weight was 8 stone 8 lbs. The temperature had never been very high, and during the last three weeks, while he had been in hospital, it had been normal. The physical signs constituted the peculiarity and interest of the case. They were as follows:—

Chest Anteriorly.—Right side moved less than left. From the clavicle to the lower border of the third rib there was slight dulness, the breath sounds were diminished, vesicular in type with no adventitious sounds, and some increase of tactile fremitus. Below the third rib there were marked dulness, bronchial breathing, increase of vocal resonance, whispering pectoriloquy, increased tactile fremitus. In the axilla the breath sounds were much diminished, and there were scattered crepitations.

Posterior Chest.—On the right side, from the apex down to within an inch of the angle of the scapula, some flatness of note and diminished breath sounds. Below this point marked dulness, bronchial breathing, increase of vocal resonance and increase of tactile fremitus. At the base of the right lung it had sometimes been possible to elicit the coin sign and at other times not. The sputum contained T. Bacilli.

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On March 25th last the patient suddenly developed complete paralysis of the left face and the left external rectus muscle. There was no pain, and there had been no recent ear trouble. The fundi were normal. There had been no improvement in this condition after three weeks' treatment by iodide of potassium. About the same time he developed marked œdema of legs and feet, with a urine of 1018 and albumen $\frac{1}{2}$. The œdema disappeared after a week in bed, but the condition of the urine had persisted, and was now 1020, albumen $\frac{1}{3}$, with a few hyaline casts.

The problems presented by this case seemed to be (1) What was the condition of the lung which gave rise to the above signs; (2) what was the cause of the albuminuria and œdema; and (3) what was the affection of the sixth and seventh left cranial nerves.

The first question that suggested itself was whether we had at the right base of this chest a localised pneumothorax or extensive cavitation. The diminished breath sounds over the upper right chest, the hollow character of the bronchial breathing, and the occasional presence of the coin sign had a little suggested this view, but it must be put aside, partly because the anterior base was dull, and partly because the tactile fremitus was increased, which it never would be with a pneumothorax. There was nothing abnormal to be found in the abdomen.

The correct reading of the condition seemed to be that the upper part of the right lung was very fibroid, and the amount of air entering it was very small, and that at the right base there happened to be several cavities quite near the surface. With regard to the albuminuria, Dr. Dawson suggested that it was due to lardaceous disease. With regard to the paralyses of the cranial nerves, the most likely explanation was that they were syphilitic. It seemed difficult, on account of the small disturbance and the good condition of the patient, to imagine how the paralyses could be caused by any tubercular deposit, but their causation was obscure.

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CASE OF PECULIAR PIGMENTATION OF THE ORAL SURFACE OF THE LOWER LIP.

By H. D. Rolleston, M.D., F.R.C.P. Lond.

The patient, a man aged 40, was admitted under my care in St. George's Hospital with abdominal pain, constipation, and some tenderness over the right iliac fossa. When the lower lip was turned down, vertical lines of black pigment were seen running in the same direction as, but superficial to, the blood vessels. Pressure on the pigmented streaks did not remove the discoloration, so, although at first sight the appearance resembled that of varicose vessels, it cannot be explained in this simple manner. The pigmentation was best marked around some raised and somewhat enlarged buccal glands, and was strictly limited to that part of the lower lip opposed to the lower incisor teeth, which were loose, very foul, and coated with much tartar. At the margin of the receding gums of these teeth there was a blackish blue line resembling a "blue line"; my colleague, Mr. H. L. Albert, who kindly saw the patient with me, was inclined to doubt whether it was a true blue line—*i.e.*, one due to lead. In the upper lip, which opposed a toothless maxilla, there was no pigmentation. There was no pigmentation elsewhere in the mouth or palate, an abnormality seen in Lascars, and there was no reason to believe that the patient was the subject of Addison's disease or hæmochromatos is.

Remarks as to the Nature of the Pigmentation.—The most plausible explanation seemed to be that the black pigmentation was due to sulphide of lead deposited in the mucous membrane, and that it was the outcome of the action of sulphuretted hydrogen, derived from the tartar around the adjacent incisor teeth, on lead carried by the capillaries to the mucous membrane. It may be mentioned that, according to Dr. T. Oliver,* the black granules of sulphide of lead, which produce the blue line, are in the deeper cells of the epidermis, and not, as Fagge taught, in the small blood vessels. There was, however, no clear history of lead poisoning. The patient was a French polisher, and to his knowledge was not exposed to the action of lead, though he admitted to drinking beer at public-houses in the early morning. Moreover, he had

* Oliver, Allbutt's 'System of Medicine,' vol. ii, p. 972.

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never had wrist-drop or gout, and though anæmic and subject to constipation and abdominal pain, had no increased arterial tension, hypertrophy of his heart, or abnormality in his urine. His urine was kindly examined chemically by Mr. F. T. Addyman, who reported that, after most careful examination by various methods, no trace of lead could be found.

There is thus no proof that the pigmentation is due to lead. Although it is difficult or impossible to exclude lead poisoning in the past as a cause of this pigmentation, it is possible that it was due to some other factor.

ULCERATION OF THE LEFT ALA NASI.

(Probably syphilitic, but unchecked by active specific treatment.)

By StClair Thomson, M.D.

The patient, a night watchman, aged 57, stated that his illness began with a running from his nose and sore throat, about 18 months ago. The swelling began on the bridge of the nose and spread downwards on the left side. He pricked the swelling with a needle, but only blood escaped. He then poulticed it, but a raw surface gradually became exposed. There was no history or trace of other specific manifestation.

On the outer side of the left ala nasi there was a circular ulcer which had perforated through into the left nasal chamber. The ulcerated surface was coated with a tenacious and very offensive slough. The edges were inflamed, and the redness extended for a short distance all round; they were not hard or tender. The glands were not affected. The patient ate and slept well, and complained of little but the local discomfort.

As the condition did not improve he was taken into the hospital, kept in bed a few days, well fed, and treated with 30 grains of iodide of potassium three times a day and inunctions of 1 drachm of unguentum hydrargyri. The ulcer was cleansed with peroxide, and dressed with iodoform and lotio nigra. Although the fœtor had been somewhat reduced, there was no arrest of the ulcerating process, and in fact it had even increased, as was shown by the fact that the base of the ala was now detached from the upper lip.

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EPITHELIOMA OF EPIGLOTTIS AND BASE OF TONGUE: TRANSHYOID PHARYNGOTOMY.

By StClair Thomson, M.D., and Albert Carless, M.S., F.R.C.S.

W.S., aged 60, was admitted to King's College Hospital, September 1st, 1900, and had been seen by Dr. StClair Thomson, at the Throat Hospital, Golden Square. The man had complained for eight or nine months of a lump at the back of his throat, which had gradually increased in size, but did not cause much pain or inconvenience. There was a large indurated ulcer with raised, everted edges, extending over the epiglottis from nearly its base, and encroaching upon the back of the tongue for nearly an inch. It was more on the left side than on the right. An enlarged and indurated gland could be felt beneath the angle of the left side of the jaw.

September 4th.—Operation. Preliminary tracheotomy. Transhyoid pharyngotomy by an incision reaching from the symphysis menti down to the thyroid cartilage; a cross cut was made in addition, but it was really unnecessary. The hyoid bone was cleared and divided in the middle line, and the two halves drawn apart. The base of the epiglottis was divided just above its attachment to the thyroid cartilage, and the pharynx opened. The growth was now easily and clearly exposed, and was removed by the scissors, cuts being made into the substance of the tongue on either side so as to remove a V-shaped portion from behind, leaving the lateral aspects of the organ, together with the vessels and nerves, intact. The wound was partially closed and the tracheotomy tube retained. The patient subsequently did very well.

October 11th.—Second operation for removal of glands which now extended down along the great vessels to which they were closely adherent.

Patient had remained perfectly well since that date, and there was now (18 months since the first operation) no sign of recurrence. He could swallow well, but his voice was a little husky, probably as the result of the occasional entrance of fluid into the larynx.

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